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THE LIFE AND ACHIEVEMENTS OF CHALMERS J. LYONS

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THE main entrance of the dental school at the University of Michigan leads to a large square reception room on the second floor. Hanging from the north wall of this room is a life-size portrait in oil. The background of the painting is subdued and, save for the head and face, the general effect is one of shadow or suggestion. Beneath the portrait and affixed to the heavy frame is a gilded plate bearing the legend, "Chalmers J. Lyons, D.D.S., D.D.Sc., F.A.C.D., 1874-1935."

Chalmers J. Lyons was born April 30, 1874, in Martinsburg, Ohio, the son of John P. and Manila White Lyons, and the third of four children. There were two older brothers and a younger sister. When Chalmers was 7, the family moved to Michigan and settled on a farm near Mount Pleasant. There, in the rural schools and at the Central State Normal College, Chalmers Lyons received his early education.

He was descended from a family of school teachers, and both his father and his mother had been teachers. At the early age of 16, he began teaching in the rural schools of Michigan. His teaching career was interrupted when he returned to the Central State Normal College to procure the necessary credits for admission to the College of Dental Surgery, University of Michigan. He entered the University in 1895 and was graduated with the degree of doctor of dental surgery in 1898.

As an undergraduate, he was far from the conception of a recluse and single-minded student; for, although well developed mentally, he was highly

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regarded by his fellow students for his affability and generosity. Among his undergraduate activities was his hobby for penmanship, mastery of which is further attested by the fact that during vacation periods he practiced it professionally. Even to one who had never known him, much could have been learned of his mind and character from his handwriting. Characteristically, it was careful, exact, simple and devoid of display. Every letter was formed and every word was clear: there was no hurry. Truly, it might be said that his handwriting revealed his character, as indeed it should, considering how much of one's mental life flows down the pen.

After his graduation, Dr. Lyons began the practice of general dentistry in Adrian, Mich. In 1907, he moved to Jackson, Mich., where he became associated in general practice with his older brother, James. In addition to conducting a busy practice, he furthered his professional proficiency by training himself in the use of porcelain, which was then coming into general use. The university dental school had been giving short courses and recognized a demand for the subject in its curriculum. Because of the prominence he had achieved in the field, Chalmers Lyons was appointed instructor in clinical dentistry, in 1907, to teach the use of porcelain. Three years later, he became a non-resident lecturer on clinical dentistry.

This contact with the university afforded him the opportunity to begin study and training in oral surgery. The major portion of the year 1908 was spent with Truman W. Brophy, of Chicago, who had pioneered in cleft lip and palate surgery and had developed a large clinic in the correction of this malady, together with other oral and maxillofacial diseases and malformations.

Upon his return from Chicago, Dr. Lyons obtained further instruction in this field by association with Drs. DeNancrede and Darling, of the surgical department of the university. At this time, oral surgery was considered in no way a specialty of either medicine or dentistry, and it was largely through his perseverance that the principles of surgery and the practice of dentistry became associated to a degree that demanded inclusion in the dental curriculum of more detailed instruction in this field. In 1913, he became instructor in oral surgery and consulting dentist to the University Hospital, this marking his complete transference to the teaching and practice of this branch of dentistry. He transferred his residence to Ann Arbor at this time and, in 1915, was made professor of oral surgery and consulting dental surgeon to the University Hospital.

With Dr. Cyrenus G. Darling, he founded the department of oral surgery in the University Hospital, in 1917. With the institution of the department, he organized an oral surgery service for the training of younger men. In the beginning, the service was limited to one year of postgraduate instruction. In 1922, it was increased to two years and, in 1923, it was lengthened to three years of clinical and technical instruction with the added optional advantage of the degree of master of science in oral surgery. At the time

of his death, at least eighteen men had availed themselves of this service and training, and by them the Chalmers J. Lyons Club was organized, in appreciation of his tutelage and for the selfish purpose of inaugurating monthly conferences with him. Shortly after his death, this group commissioned a portrait painter to execute the likeness that hangs in the dental school, and presented it to the University, in 1936.

Dr. Lyons' influence upon his pupils was profound. He was especially endowed by heredity, environment, education and temperament to become a great teacher. In both the dental and the medical school, he impressed his students with his simple, dignified and thorough teaching methods. His words were carefully chosen and deliberately uttered and he rarely failed to make his point. He carried his students along with him, and perhaps no teacher's words were ever better remembered. Earnestness was his eloquence, and a desire to instruct, his motive power. There was that about him which engendered seriousness and his students quickly became infected by his attitude. His influence as a teacher extended far beyond the classroom. Unconsciously, he taught the finer tenets of life by exemplifying them in his daily existence.

As a member of the faculty and an advisor in matters of policy pertaining to the school, his counsel was invaluable. He was vitally interested in the affairs of the dental school. The development of a curriculum to meet the changing conditions of dental education and the organization of postgraduate teaching and the various departments of undergraduate instruction were matters of great importance to him. Because of the honesty of his purpose and the clearness of his judgment, which were recognized by all who knew him, his assistance was frequently sought by the dean and other members of the faculty in the solution of many difficult problems.

He was unquestionably the outstanding figure of the dental faculty. Other professors were eminent and many of them left their mark in their work, but Dr. Lyons' striking personality, embodied in a frame of manly proportions, his expressive features suffused with a tenderness peculiarly his own, combined to inspire confidence and endear him to all those who were associated with him.

When, in 1934, he was made chairman of the executive committee of the school of dentistry and additional administrative duties were thrust upon him, he showed great tact and skill in meeting the many difficult situations that arose in the performance of his official duties.

His relations with patients were characterized by the fact that he never allowed the professional problem to submerge the personal equation. Patients were patients and not cases. There was always great charity for the weakness of human nature and there were neither unkind nor hasty judgments. He never allowed his poise to be disturbed or his expression to show what would be better concealed.

There was one subject which he never discussed with a patient, and that

was the possible shortcomings of another dentist or physician. At such inferences, he showed his displeasure at once, and if this were not enough, he either issued a sharp rebuke or assured the patient regarding the doctor's ability. In fact, this was about the only thing that caused him to lose patience and was the rare occasion of his showing sternness.

Of the attitude of patients toward him, perhaps the most striking characteristic was absolute confidence. There was the certainty that there would be no lack of skill or interest on his part. In giving an opinion, he put matters simply, so that the chance of misunderstanding would be as slight as possible. He was careful to avoid saying anything in the hearing of a patient that might cause disturbance or increased anxiety. In giving advice, he was sparing of words and might be described as one of those "who have not the infirmity, but the virtue of taciturnity, and speak not out of the abundance, but the well-weighed thoughts of their hearts."

In the field of oral and maxillofacial surgery, he developed a national and international reputation. His cleft lip and palate clinic at the University Hospital grew to be one of the largest in the world, and surgeons from the four corners of the earth were in almost constant attendance at his surgical clinics. He was greatly in demand as a lecturer and clinician throughout the United States and abroad. In 1925, he visited the surgical clinics of Europe and returned greatly impressed with the superior type of work being done in this country in the field of oral and maxillofacial surgery.

He was a frequent contributor to dental and medical literature. He wrote a textbook on "Fractures and Dislocations of the Jaw" and he contributed chapters dealing with oral surgery to Ward's "American Textbook of Operative Dentistry" and in Mead's "Oral Surgery." Unity, order, clarity of description and ease of diction mark his textbooks and his various monographs and other writings. The information is imparted in such a manner that the reader at no time finds it difficult, because of ambiguous phraseology or doubtful expression, to grasp his meaning.

Despite his busy life, Dr. Lyons participated actively in many professional and civic organizations. He was a member of the American Medical Association, American Dental Association, American Association of Oral and Plastic Surgeons and International Association for Dental Research and a Fellow of the American College of Dentists and was on the Michigan State Advisory Council of Health. He was a member of Sigma Xi, Phi Kappa Phi, Omicron Kappa Upsilon and Delta Sigma Delta fraternities. He served as president of the American Association of Oral and Plastic Surgeons and the Michigan State Dental Society, also as Supreme Grand Master of Delta Sigma Delta fraternity. In 1933, he was awarded the Jarvie Medal given by the New York State Dental Society for outstanding dental service.

Dr. Lyons was twice married. A son, Richard Hugh, born of the second marriage, is now assistant professor of medicine at the University of Michigan School of Medicine.

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Despite his rugged physique, Dr. Lyons' many activities were interrupted by illnesses during the last several years of his life. Three major operations had had their effect and his health was declining, but he remained active until on the morning of May 14, 1935, when he was suddenly taken acutely ill with what proved to be a perforating gastric ulcer. Although an early operation was performed, he succumbed three days afterward.

It is granted to few men to see the fruits of their labors. Usually, it is left to posterity to recognize their worth and raise monuments in their honor. Also, each generation may be inclined to forget the labors of those who went before it and whose labors contributed so largely to its own strength. Our own generation of dentists, however, can never forget that it was Dr. Lyons who built so well and contributed so much to dentistry, and particularly to oral surgery. For his handiwork and his vision, many hundreds of dentists living today are better men and better practitioners, having had the benefits of his instruction, and thousands upon thousands of the sons of men have been happier since he lived, because of the chapter he wrote in the book of human life, where, as Osler said, "are set down the glad tidings of the final conquest of nature by which man has redeemed thousands of his fellowmen from sickness and from death."

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SIGNIFICANCE OF BLOOD DYSCRASIA IN ORAL SURGERY

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THERE is considerable evidence in the literature that a large percentage of patients with blood dyscrasia make their first professional contact with their dentist. This is primarily because many of the blood diseases have early manifestations in the oral cavity; either producing oral lesions or, by their hemorrhagic tendencies, causing vicious bleeding after extraction of teeth. Forkner[1] reports that all six of his patients having monocytic leukemia had consulted a dentist because of gingivitis, in the early stages of illness. Mercer[2] found that dental treatment had been carried out in both of his cases of the disease. Comroe,[3] Kracke,[4] Cook[5] and Epstein[6] all emphasize the early appearance of oral lesions and stress the importance of early diagnosis.

It is not to be supposed that the oral lesions alone are diagnostic of the disease. However, the picture is generally clear enough to arouse suspicion that a blood disease is the etiologic agent. In many instances, an accurate provisional diagnosis can be made from the oral lesions plus the history and physical appearance of the patient. However, it is the blood studies that are diagnostic, and they should be made when even the slightest suspicion is aroused. Certainly the smooth, red, tender tongue of pernicious anemia (Hunter's glossitis), the necrotic gum tissue of agranulocytic angina, the hemorrhagic hypertrophied gum tissues of scurvy, the insidious steady bleeding of purpura hemorrhagica, etc., should arouse the feeling that all is not well and that the lesions are not purely local.

A blood dyscrasia is, by definition, a disease of the blood and blood-forming organs. Therefore, the blood may, or may not, be the only structure involved. The disease may be associated with marked enlargement of the spleen, liver and lymph nodes, or there may be a hypoplasia or aplasia of the bone marrow.

The blood is composed of a colorless liquid (plasma) in which float various types of cells. The accepted normal count for these cells is as follows: red blood cells: adult males, 5,500,000 per cubic millimeter; adult females, 4,800,000 per cubic millimeter; hemoglobin content (100 per cent), generally 15.6 gm. per hundred cubic centimeters; white blood cells, 7,000 per cubic millimeter: polymorphonuclear neutrophils, 60 per cent; eosinophils, from 2 to 4 per cent; basophils, 1 per cent; lymphocytes, from 20 to 30 per cent, and monocytes, from 4 to 8 per cent.

These counts being considered normal, any appreciable increase or de-

From the Department of Oral Surgery, Henry Ford Hospital.

crease can be considered as pathologic. The change may be transitory, as in the leukocytosis seen in gross suppurative infections, and is largely a defense mechanism of the body to phagocytize the invading bacteria.

The oral manifestations of a blood dyscrasia can be placed in two broad classes: (1) lesions of the oral mucous membrane and (2) oral hemorrhage without demonstrable mucous membrane lesions.

There is some overlapping of these two classes since many of the blood diseases that show demonstrable oral lesions also have a marked hemorrhagic tendency.

Class 1 includes anemia: pernicious, hypochromic and aplastic; leukemia (acute and chronic): lymphatic, myelogenous and monocytic; agranulocytic angina; purpura; polycythemia vera, and scurvy.

Fig. 1.—Hunter's glossitis of pernicious anemia. The tongue is bright red and there is a generalized atrophy of the papillae. The lesion, similar to that found in vitamin B deficiency, clears up promptly with dietary adjustment and liver extract therapy.

Class 2 includes hemophilia, vitamin K deficiency, vitamin C deficiency (without oral scurvy) and purpura, in which there may be no oral lesions.

ANEMIA

The common outstanding oral sign of all types of anemia is a generalized pallor of all the mucous membranes, due to the decrease in the hemoglobin content of the circulatory blood. In pernicious anemia, this may also be associated with Hunter's glossitis in a large percentage of cases. This is productive of a red, tender, bald condition of the tongue, which may be completely devoid of papillae. (Fig. 1.) The condition is probably associated with a vitamin B deficiency, since all of the cases show a persistent

achlorhydria and the tongue becomes normal again with adequate liver therapy. Certainly, any patient presenting himself for dental examination and complaining of a sore tongue should have the benefit of a complete blood count. The glossitis present in some cases of hypochromic anemia is quite different from Hunter's glossitis. It is patchy, various areas being devoid of papillae, and there may be small petechiae in the otherwise pale oral mucosa.

Aplastic anemia, having a poorer prognosis than either pernicious or hypochromic anemia, owing to a deficiency in or absence of blood regeneration, rarely has a glossitis associated with it. It instead causes a marked tendency to hemorrhage, spontaneous or resulting from minor trauma, and a metallic line may be present in the gum tissues, being due to arsenical intoxication.

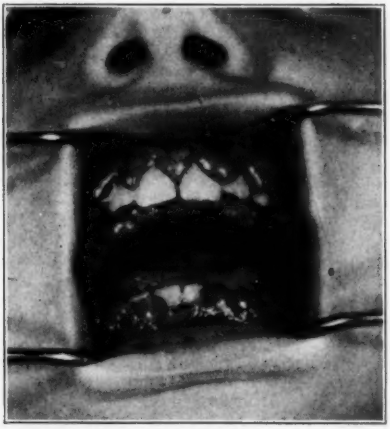

Fig. 2.—Acute stomatitis of monocytic leukemia. The gingival hypertrophy was most pronounced in the lower anterior jaw where the teeth were almost completely hidden from view. These lesions were accompanied by a persistent necrotic ulcer in the lower right molar area. The teeth were loosened and there was continual oozing of blood. The patient came originally to the Oral Surgery Outpatient Clinic complaining of sore mouth of one week's duration. The temperature was 101.2 F. On admission to hospital, the provisional diagnosis was leukemia. A complete blood count revealed a monocytic leukemia with a total white cell count of 17,400, of which 83 per cent were monocytes. The disease ran its course in sixty-four days, terminating fatally.

The blood pictures of the three above-described anemias are diagnostic. The color index (hemoglobin percentage divided by the percentage of normal that the red blood cells represent) is always more than 1 in pernicious anemia, while in all others it is always less than 1. Hypochromic anemia shows a characteristic depression in the hemoglobin content in combination with a relatively normal red blood cell count; whereas, in aplastic

anemia, there is a relatively equal depression of the red blood cells and hemoglobin and an associated leukopenia and reduction in platelets.

LEUKEMIA

Leukemia is a progressively fatal disease characterized by the production of an excessive number of immature white blood cells. In advanced stages of the disease, few adult cells are present. Since the immature white blood cells do not have the ability to combat infection to the same degree, gross infection becomes evident and overwhelming. Even in the early stages of the disease (especially in monocytic leukemia), necrotic ulcers may form

Fig. 3.—Gangrenous stomatitis in an advanced myelogenous leukemia. The patient was seen originally in the emergency room of the hospital with a complaint of persistent hemorrhage following extraction of several teeth. The only discernible lesion at that time was a severe hypertrophic gingivitis. Necrosis began shortly after admission to the hospital. Death ensued on the eighteenth day in hospital.

in the mouth, this possibly being the first symptom to present itself. There is a marked tendency to gingival hyperplasia and the gum tissues may almost conceal the teeth. (Fig. 2.) The gum tissues are a deep, intense red and tend to fall away from the teeth. Spontaneous hemorrhage from the gum tissues may be quite frequent.

Leukemia may be acute or chronic, the ultimate prognosis always being hopeless.

All three forms, lymphatic, myelogenous and monocytic, of acute leu-

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kemia present somewhat the same oral picture except, as has been noted by some authors,[1, 2] early appearance of marked stomatitis gingivitis, ulceration and necrosis, bleeding and hyperplasia of the gum tissues are more frequently present in the monocytic type. In any event, the appearance of marked gingival hyperplasia, with deep redness or without accompanying necrosis in a child or young adult giving the history of an acute onset accompanied by fever and pain, should immediately place the examining dentist on guard. The only difficulty in differential diagnosis might lie in the possibility of acute Vincent's infection. As a result, any case of Vincent's infection that does not immediately show improvement should be viewed with suspicion and a complete blood count, including differential white blood cell count and hemoglobin determination, should be made.

The total number of leukocytes may vary from 10,000 to 250,000, 50

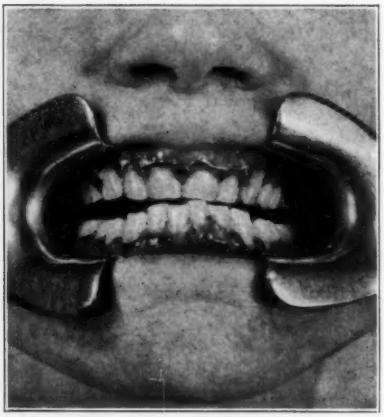

Fig. 4.—Typical gingivitis of scurvy. The gums are engorged and edematous. There is often slow oozing of blood from the gingival margins. It can be seen here in the upper right cuspid area. A combination of vitamin C therapy and repeated oral prophylaxis is necessary to entirely clear up the oral lesions.

per cent or more being immature forms. The differential white blood count reveals the type of leukemia. There is generally an accompanying progressive anemia, which tends to become severe unless checked by transfusion.

Oral treatment is limited to the use of mild oxidizing agents and saline irrigations. Daily application of 1 per cent gentian violet seems to make the patient more comfortable.

As in acute leukemia, the chronic forms present oral symptoms that are very similar, but unlike the acute type unless the patient has an exacerbation of the disease. The chronic forms are generally encountered in persons over 35 and may be present in one who appears in chronic ill health or one

TABLE 1.—TYPES OF BLOOD DYSCRASIA THAT PRODUCE ORAL LESIONS.

	Blood Findings	Oral Findings	Physical Signs	Prognosis
I. Anemia				
Pernicious	Low red cell count Low hemoglobin Color index 1+	Hunter's glossitis Pale mucous membrane	Lemon-yellow skin tint Weakness Tingling of extremities	Excellent
Aplastic	Low red cell count Low hemoglobin Color index 1 or 1— Progessive leukopenia Reduction of platelets	Pale oral mucosa Possible metallic deposit in gums Tendency to spontaneous hemorrhage	Progressive weakness Pallor	Poor
Hypochromic	Normal red cell count Low hemoglobin Color index less than 1	Patchy glossitis Pale mucosa Small petechiae	Pallor Chronic ill health	Good
II. Acute leukemia				
Monocytic	Anemia Leukocytosis with high percentage of monocytes Decreased platelets	Marked tendency to hyperplasia of gum tissues, ulceration and necrosis Severe pain	High temperature Acute onset Early onset Pallor Prostration	Hopeless
Lymphatic	Anemia Marked leukocytosis with high percentage of immature lymphocytes	Similar in all acute leukemia except tendency to greater involvement in monocytic type	Same as in monocytic type	Hopeless
Myelogenous	Similar to lymphatic leukemia except difference in staining characteristics	Same as above	Same as above	Hopeless
III. Chronic leukemia				
Lymphatic	Anemia Leukocyte increase generally below 100,000 per cu. mm. 90-95% of white blood cells are small lymphocytes Reduced platelet count	Chronic stomatitis with hyperplasia of gingival tissues; loosening of teeth; tendency to spontaneous hemorrhage; possible necrosis and gangrene.	Weakness Fatigue and pallor Swelling of lymph nodes Cough Chronic pruritus	Poor Life expectancy up to five years

(Continued on next page)

TABLE 1.—*(Continued)*

	Blood Findings	Oral Findings	Physical Signs	Prognosis
Monocytic	Anemia Leukocytosis (may or may not be severe) with high percentage of monocytes	Added tendency to ulcerative stomatitis	Weakness Pallor Weight loss May or may not have lymph node enlargement	Poor
Agranulocytic Angina	Sharp decrease in white cells, especially affecting granulocytes	Necrotic gingivitis and stomatitis Ulcerative pharyngitis May follow dental extraction or use of aminopyrine-containing compounds Found frequently in physicians, dentists and nurses and their relatives	Acute onset with fever and prostration Must differentiate from severe Vincent's infection by blood count	Hopeless in 50 to 95% of cases
Purpura	Normal blood count Normal coagulation time Decreased platelet count Delayed clot retraction Positive tourniquet test	Petechiae in buccal mucous membrane Spontaneous hemorrhage Severe hemorrhage after extractions	Flesh bruises easily Multiple large ecchymotic areas on extremities	Generally good, but may be hopeless

TABLE 2.—TYPES OF BLOOD DYSCRASIA THAT MAY CAUSE HEMORRHAGE.

	Blood Findings	Oral Findings	Physical Signs	Prognosis
Hemophilia	Normal bleeding time, platelet count and clot retraction Prolonged clotting time Probable anemia due to blood loss	Marked uncontrollable hemorrhage after extraction or other operation	Generally a history of hemophilia	Poor
Polycythemia vera	Marked increase in red cells and hemoglobin percentage 8,000,000 to 9,000,000 red blood count and hemoglobin up to 150% not unusual	Reddish purple hue of mucosa Prolonged hemorrhage	Reddish purple hue of skin Enlargement of spleen Vertigo	Life expectancy about eight to ten years
Vitamin C	May be decrease in platelets Low or absent vitamin C blood content	Generally have swollen hemorrhagic gums	Ecchymotic areas in skin History of no intake of citrus fruits or tomato juice	Good

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Vitamin K	Decreased blood prothrombin level Lengthened coagulation time May have anemia from hemorrhage	No lesions	Hemorrhagic disease of newborn Often occurs in jaundiced persons, being due to lack of bile in gastrointestinal tract

TABLE 3.—INDICATIONS FOR BLOOD STUDIES.

Oral Findings	Rule Out	Blood Studies Necessary
Persistent necrotic stomatitis	Leukemia Diabetes Scurvy	Complete blood count and differential Stained blood smears Platelets, vitamin C level. If negative, blood sugar is shown
Acute ulcerative stomatitis	Acute leukemia Agranulocytic angina Vincent's infection	Complete blood count including hemoglobin and differential white cell count Stained blood smears
Chronic, diffuse, gingivitis associated with bleeding from gum tissues	Chronic leukemia Vitamin C deficiency History of epilepsy and treatment with dilantin	Complete blood count Hemoglobin determination and differential white cell count Stained blood smears Vitamin C blood level
Sore, red, bald tongue with or without pallor	Pernicious anemia Vitamin B deficiency	Complete blood count including hemoglobin and differential white cell count Physical examination
Petechiae in oral mucosa Ecchymotic areas in skin History of bleeding	Aplastic anemia Pernicious anemia Purpura Polycythemia Hemophilia Vitamin C deficiency Vitamin K deficiency	Complete blood count including hemoglobin and differential white cell count Bleeding and coagulation time, clot retraction Platelets Prothrombin Blood vitamin C
History of hemorrhage	Hemophilia in males Purpura Vitamin C deficiency Vitamin K deficiency Polycythemia Leukemia Aplastic anemia (look for metallic line)	Complete blood count Bleeding and coagulation time Clot retraction Platelets Prothrombin Vitamin C

who shows no outward signs of illness. The outstanding oral symptom is the presence of an acute or a chronic gingivitis. The gum tissues are typically engorged and swollen and are a deep red. The involved teeth tend to become loose and sore and unprovoked bleeding is to be expected. Extraction of teeth is definitely contraindicated unless it can be carried out during a remission of the disease. Relatives of the patient should be advised that extraction may be followed by severe bleeding and an acute exacerbation of the disease.

The patient should be hospitalized and should have adequate medical supervision. Transfusion before and after extraction is sometimes necessary.

Although the oral and physical findings do not form a basis for differential diagnosis among the various forms, the blood picture is striking and diagnostic. Chronic myelogenous leukemia causes a marked leukocytosis with a white blood cell count as high as 1,000,000 per cubic millimeter, with a high percentage of immature myelocytes. In chronic lymphatic leukemia, the white blood cell count is generally below 100,000 per cubic millimeter, with from 90 to 95 per cent small lymphocytes. There is an accompanying reduction in the platelet count. The leukocytosis of chronic monocytic leukemia may not be severe, but there are a high percentage of monocytes present.

AGRANULOCYTIC ANGINA

Agranulocytic angina is considered a relatively new clinical entity, first described by Schultz[7] in 1922. It is characterized by progressive prostration, fever, formation of necrotic ulcers in the gingival, buccal and pharyngeal areas and a marked depression of the white blood count with few or no polymorphonuclear leukocytes present. The ulcers, which may be very deep and extensive, are accompanied by severe pain, *fetor oris* and enlargement of the regional lymph nodes.

The disease appears most frequently in physicians, dentists and nurses and their relatives. Since aminopyrine (amidopyrine, pyramidon) seems to be the most universally accepted etiologic agent, it is strongly suspected that the prolific free distribution of aminopyrine-containing anodynes to the professions is a definite factor in the disease. Present evidence points to the conclusion that the disease is based on individual idiosyncrasy to the drug.

Dental extractions also seem to be a factor. Schaeffer[8] has reported eighteen cases following dental extraction with a mortality rate of 90 per cent. He was unable to ascertain whether any of the patients had used aminopyrine or aminopyrine-containing compounds postoperatively.

The disease runs a typically acute course with a fatal outcome in a high percentage of cases, in from two to fifty days. As yet, there is no specific form of therapy available, but the best results have been obtained by the vigorous use of pentose nucleotides, liver extract and leukocyte cream.

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PURPURA HEMORRHAGICA

Purpura hemorrhagica is characterized by spontaneous hemorrhage into the skin and mucous membranes, giving rise to the so-called purpuric spots, which are commonly found in the extremities. The patient will rarely recall any trauma preceding their formation. Spontaneous nose bleed is typical. True thrombocytopenic purpura is associated with a marked decrease from the normal platelet count of 250,000 per cubic millimeter, with an accompanying decrease in the prothrombin content of the blood. Bleeding time is prolonged, coagulation time is normal. Clot retraction is delayed or absent.

Mild chronic cases may be greatly improved by elimination of focal infection, as reported by Pemberton.[9] In severe cases, it may be necessary to resort to splenectomy during a recession of the disease to obtain more lasting improvement. Ligation of the splenic vessels is occasionally carried out during an acute stage of the disease as a life saving measure. It is followed by splenectomy at a later date, if the course of the disease warrants it.

Extraction or any other oral surgical procedure is certainly contraindicated during the acute phase of the disease. If it is the chronic type, elimination of severe focal infection may be necessary to obtain improvement. All precautions to guard against hemorrhage must be taken and the bleeding and coagulation times must not be unduly high.

SCURVY

Scurvy, which used to be widespread and is now comparatively rare, is based on a diet deficient in vitamin C. The oral lesions may precede all other signs of the disease or they may be entirely absent. The lesions are characterized by a granulomatous hypertrophy of the gum tissues, which bleed on the slightest trauma. Because of secondary infection, the oral lesions will not entirely disappear with heavy doses of cevitamic acid. A series of oral prophylactic treatments is necessary unless the involvement has been sufficiently destructive to warrant extraction of all remaining teeth.

Oral lesions, as mentioned above, may be entirely absent. A low vitamin C blood content, which should always be considered a possible factor in any unexplained bleeding, is occasionally present in postoperative hemorrhage. The mechanism of bleeding in vitamin C deficiency is associated with a capillary fragility due to a lack of collagenous material in the fibrous tissue and diminished cohesion of the endothelial cells.

If the blood vitamin C content is below 0.3 mg. per hundred cubic centimeters, heavy doses of cevitamic acid (100 mg. three times daily) should be given in an attempt to raise the level to 1.0 mg. per hundred cubic centimeters. A product for intravenous injection is available for use when necessary. Ordinarily, the oral administration is adequate and satisfactory.

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HEMOPHILIA

Hemophilia is a strictly hereditary disease. The diagnosis is generally established early in life, well before any surgical measures are necessary. It is a constitutional derangement and prolonged bleeding time with normal clotting time is observed. There is also a decrease in the prothrombin content and an increase in the antithrombin percentage in the blood.

Extraction of teeth becomes almost mandatory in some cases of hemophilia. This has been the cause of much distress for both the patient and the dentist. Recent research[10] has given encouraging results from the local application of Russell's viper venom to the bleeding area. A dilution of 1:10,000 of sterile venom has been used with good results.

Certainly a patient of this type should be hospitalized, and adequate blood of the correct type should be available to make up for the loss that is to be anticipated. Every effort should also be expended to correct the dental pathosis without resort to extraction.

VITAMIN K DEFICIENCY

Vitamin K has been used as a specific in treatment of hemorrhagic disease of the newborn. It is also applicable in all cases of bleeding associated with obstructive jaundice and in other cases of bleeding when all other bleeding factors are normal except for a reduction of the prothrombin content and a lengthened coagulation time. When jaundice is present, it is necessary to administer bile salts along with the vitamin K to allow for its absorption.

The vitamin can be given empirically in an emergency and is very effective on occasions. However, it is a much sounder policy to test the bleeding factors prior to its administration.

SUMMARY

Since the various types of blood dyscrasia frequently produce oral lesions as the first demonstrable symptom, the dental profession should be sufficiently familiar with the disease to advise blood studies and physical examination in cases warranting them.

It must be kept in mind that most of the types of dyscrasia that produce oral lesions also predispose the patient to dangerous hemorrhage.

Tables 1 and 2 present the salient diagnostic features of each disease. Table 3 brings out the possible diagnoses of the various oral symptoms, together with the diagnostic studies necessary.

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Diagnosis.—One of the weakest links in the practice of medicine or dentistry is diagnosis. I like this definition: "Diagnosis is the conclusions arrived at in the process of applying the mind to a collection of scientific data." All too often, the practitioners of either medicine or dentistry fail to take into consideration a collection of scientific data, but are content to base their decisions upon one or two outstanding objective signs. Of course, the large majority of patients that the dentist sees are ambulatory, but in these patients there is a difference in local and general systemic conditions that the dentist can easily overlook; which does not always work out for the best interest of either the dentist or the patient. It must be obvious to all that in the case of many patients who come to the dentist for certain definite relief, only a cursory examination is required in order to determine the operative procedure. It is not toward this type of patient that this discussion will be directed, but to a large group of patients who daily come to the dental office with either a local or a systemic disturbance and who require a careful analysis of all possible factors in their condition in order that the character of the operative procedure may be determined. In many of these cases, a complete history of the patient is necessary in order that a judicial decision may be rendered. In other words, we must be able to see the individual rather than just the local condition. Dentistry has accepted the guardianship of the mouth and has assumed the responsibility of keeping it in a condition that will be in harmony with bodily health. Years of scientific research have made it plain that the effect of oral conditions depends very largely on the degree of resistance of the individual. Much controversy in both the medical and the dental profession relative to the subject of focal infection would have been dissipated long ago had this principle been recognized early.—Chalmers J. Lyons, in *J.A.D.A.*, 1932.

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PROBLEMS OF TOXICITY IN SULFONAMIDE THERAPY

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ALTHOUGH sulfonamide drugs are of great therapeutic value, the numerous reports of toxic effects of the use of these compounds are evidence that the drugs are dangerous as well as life-saving. The danger lies not only in the immediate toxic effects, but also in remote effects, which may be even more serious. With the development of new compounds from the sulfanilamide base, the immediate toxic effects have been considerably decreased. Consequently, there is a greater tendency to regard the occasional toxic effect as of minor significance and to administer the drugs to patients in whom there may be only a remote possibility that such therapy will be helpful. Too little consideration is often given to the possibility of producing a sensitivity to the drug that may make the subsequent use of sulfonamide compounds dangerous or contraindicated when the patient is suffering from an infection for which sulfonamide therapy might be life-saving.

It is difficult to evaluate the incidence of the toxic effects from the reports in the literature since this will vary with the type and amount of the drug administered, and with the duration and repetition of treatment. Thus the number of cases exhibiting toxic reactions after receiving sulfonamide therapy for two or three days will be considerably less than if therapy were continued for twenty or thirty days. Long,[1] in a summary of the toxic effects of the commonly used sulfonamides given to several thousand patients in variable doses over variable periods, has found that the incidence of toxic manifestations, such as fever, rash, hemolytic anemia, leukopenia, agranulocytosis, hematuria, oliguria and hepatitis, was 11.9 per cent with sulfanilamide therapy, 15.9 per cent with sulfapyridine, 18.6 per cent with sulfathiazole and 6.5 per cent with sulfadiazine. The experience with sulfamerazine or sulfamethazine is as yet insufficient to permit clear-cut conclusions concerning the incidence of toxicity of these drugs. The reports at the present time, however, would suggest that sulfamerazine has about the same toxicity as sulfadiazine.[2]

The untoward reactions common to all sulfonamide compounds may, for the sake of simplicity, be roughly classified into three groups: a direct toxic effect of the drug, which may manifest itself shortly after the drug

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has been administered; a sensitization to the drug, which may appear after several days of continued therapy or after the readministration of the drug, and the effects on the kidneys and urinary tract from the precipitation of the drug or its acetylated form. Unfortunately, the information concerning some toxic manifestations is insufficient at present to permit a clear-cut classification of them into one of the three above-described groups. This is particularly true of the blood disorders that follow the use of the sulfonamides, since some reactions might suggest a direct toxic effect, while others with similar clinical characteristics would appear to be the result of a sensitivity.

The immediate toxic effects of the drug are usually mild, but may vary in intensity and frequency with the dose and type of sulfonamide compound administered. In susceptible patients, administration of any of the compounds will produce nausea and vomiting, presumably through a direct toxic effect on the central nervous system. These symptoms were common with sulfanilamide and sulfapyridine, but occur in only about 10 per cent of cases treated with sulfathiazole, sulfadiazine and sulfamerazine. The other effects on the central nervous system are noted less commonly with the last three drugs than they were with the earlier compounds. Headache, dizziness, lack of coordination, mental lapses and psychoses, though not uncommon sequelae of sulfanilamide and sulfapyridine therapy, occur so rarely with sulfathiazole or sulfadiazine that these drugs may be given in small doses to patients pursuing their usual daily routine. At times, however, serious nervous system disturbances will occur from these drugs, especially when administered in large doses or to patients with preexisting disease of the nervous system. Dysmorphophobia, aphasia, agraphia, stammering, toxic psychosis, peripheral neuritis, encephalomyelitis, myelitis, optic neuritis, transitory myopia, meningeal signs, blindness and convulsions all have been described as resulting from the use of sulfonamides.[3]

Some reports suggest that the sulfonamides may have a direct nephrotoxic action as well as produce lesions in the kidney as a result of precipitation of the drug. In such cases, there is usually evidence of renal involvement early in the course of therapy, consisting of severe albuminuria, oliguria, anuria and nitrogen retention associated with microscopic evidence of tubular degeneration, glomerular swelling and occasionally widespread necrosis of the renal parenchyma.[4]

HYPERSENSITIVITY REACTIONS

One of the most common and serious complications of sulfonamide therapy is the hypersensitivity reaction. These reactions are in many respects analogous to the clinical syndrome associated with serum sickness. As with serum sickness, the sulfonamide reaction is likely to develop in the second week with continued administration of the drug and is usually associated with many or all of the following symptoms: fever, chills, nausea and vom-

iting, skin eruption, conjunctivitis, muscular aches, arthralgia, profound prostration often out of proportion to the fever, and transient delirium.[5] At times, jaundice, oliguria, anuria, edema, lymphadenopathy and leukemoid reactions have also been associated with this type of reaction. It has been demonstrated that sulfonamides may combine with plasma protein.[6] It is very likely that, in the body, a combination of protein and the sulfonamide compound may be formed which will act as a foreign protein to which an immunologic reaction is established similar to that associated with the injection of foreign serum. It differs, however, from serum sickness in that no antibodies to the sulfonamides have been found and the usual efforts to demonstrate sensitization have in general been unsuccessful. Wedum,[7] on the other hand, has been able to demonstrate sensitivity reactions by skin tests, the use of precipitins and anaphylactic shock in guinea-pigs given sulfonamide azoprotein.

Since this type of reaction rarely occurs before the seventh day, and at times not until the third week of therapy, many persons receiving sulfonamides for a shorter time will not exhibit these symptoms, but may, nevertheless, develop a sensitivity that is not manifest because of the discontinuation of therapy. It is nearly impossible to estimate the incidence of such a state of sensitivity produced in this manner since no symptoms may develop with an initial course and little attention has been paid to the minor symptoms of malaise, muscular ache and nausea and vomiting occurring after several days of treatment. The incidence of the more dramatic symptoms, such as fever, chills and dermatitis, has been determined by many observers. According to Long's large collection of cases, fever and dermatitis have been found in 5 and 2.2 per cent, respectively, of the cases treated with sulfanilamide; 3.1 and 2 per cent with sulfapyridine; 6 and 5.2 per cent with sulfathiazole, and 1.6 and 1.3 per cent with sulfadiazine.[1] From the few reports concerning sulfamerazine, it appears that about 3 per cent of patients developed fever and 2-3 per cent developed dermatitis. These figures express only the incidence of two types of sensitivity reactions noted in routine hospital practice. It is very likely that the incidence of sensitivity will be considerably greater if it is judged by the response to readministration of the drug shortly after completion of an initial course of therapy, since, in many cases, sulfonamide therapy is discontinued before sufficient time has elapsed for a hypersensitivity reaction.

In an effort to determine the incidence of sensitivity, sulfathiazole was readministered to fifty-three patients, from two to fourteen days after completion of an asymptomatic first course. Nineteen of these patients (36 per cent) developed a severe febrile reaction, usually within a few hours after the readministration of the drug.[5] From this, it seems that readministration of the drug shortly after completion of an initial course of therapy increased the incidence of hypersensitivity reactions above that found with continued therapy. It appears probable that if a longer period of time

elapsed before readministration of the drug, fewer reactions might be expected. The incidence of such sensitivity reactions to sulfadiazine therapy in adults does not seem to be so high, since these reactions occurred on readministration of the drug in only three of thirty-four patients that received the initial course without difficulty.[8]

Fink and Wilson,[9] in a study of the readministration of sulfathiazole in eighty-six children and sulfadiazine in ninety-one children, found that only 4 per cent of the 177 patients developed fever on the second course of therapy. The interval between the first and second courses varied from one day to one and one-half years. All seven patients in whom fever developed had finished the first course of the drug from one to six weeks before development of fever with the second course. The febrile reaction in these cases did not occur immediately with readministration of the drug; which suggested that it had no clear relation to previous drug therapy. This would suggest also that sensitization to the sulfonamides is uncommon in children.

When a reaction associated with hypersensitivity to the drug does occur, readministration of the drug at any time in the future is likely to have the reaction. In the febrile reactions, readministration of the drug will again produce fever in about 75 per cent of the cases.[1, 5] Such a state of sensitivity has been found to persist for as long as two years.[10] There is often considerable specificity in the type of sensitivity produced. Many patients who show a hypersensitivity reaction to one sulfonamide compound may be able to take another compound without difficulty.[5] This is not always true. A state of hypersensitivity to all sulfonamides or another compound may be present.

Fever, which is one of the striking symptoms of sulfonamide hypersensitivity, usually develops during the second or third week of continued drug therapy. It may start insidiously and gradually increase in severity as the drug is continued or come on abruptly with shaking chills and a rise to 105-106° F. in a few hours. In some cases, fever has developed after the drug has been discontinued.[11] In general, with the prompt cessation of sulfonamide therapy, the fever begins to subside gradually, though some temperature elevation may persist for several days after such an episode. The differentiation between fever resulting from the drug or from the disease for which the drug is administered is at times difficult. Other manifestations of the sensitivity reaction, such as skin eruptions, conjunctivitis, severe prostration, arthralgia and the time interval from the onset of therapy, are helpful in making such a differentiation. The presence or absence of leukocytosis will not be of aid. Though the majority of the cases with a hypersensitivity reaction fail to show significant changes in the blood picture, others may show considerable changes and at times a leukemoid response.

Intermittent administration of the sulfonamides is particularly apt to have hypersensitivity reactions. Readministration of one of the compounds

a week or more after the initial exposure to the drug may be associated with an abrupt rise in temperature and the other symptoms of sensitivity. The severity of the reaction under the circumstances may be in part determined by the amount of the drug administered, the time interval between courses and the state of sensitivity.

Skin rashes of many types have been noted as following sulfonamide administration, like fever usually developing during the second or third week of therapy. Skin changes, though often associated with fever, may occur with other manifestations of hypersensitivity. The type of dermatitis varies considerably from patient to patient and depends to some extent on the type of sulfonamide compound used. An erythema nodosum or an urticarial dermatitis is more commonly seen after administration of sulfathiazole, while a morbilliform or maculopapular type of reaction is more often seen with sulfadiazine.

The skin appears to be somewhat photosensitive and the eruption is more likely to follow exposure to sunlight. In most cases, the dermatitis will gradually subside when the sulfonamide therapy is stopped. The continued administration of the offending drug tends to increase the severity of the eruption and the other manifestations of the hypersensitivity reaction may then become more pronounced. In some cases, continued therapy has led to the development of serious and sometimes fatal exfoliative dermatitis. Readministration of the drug is likely to cause a second eruption of the skin.

It is interesting that the local application of sulfathiazole ointments may be associated with sufficient absorption of the drug to cause not only a local cutaneous sensitization,[12] but also a general hypersensitivity to the drug when it is administered orally.[13] The cutaneous reaction may take the form of a local contact type of dermatitis, with or without a disseminated eruption, or it may appear as a local or generalized exacerbation of the dermatitis for which the patient is being treated. (Schaeffer.[12])

The oral administration of sulfathiazole to patients who have used sulfathiazole ointment for some time in the past has resulted in not only manifestations of a generalized hypersensitivity, including fever, chills and malaise, but also a dermatitis, often localized in the areas previously exposed to the drug.[13] Evidence of the cutaneous sensitivity can at times be obtained by patch tests. Somewhat similar reactions have been described in a patient sensitive to procaine and other local anesthetics who had erythematous pruritic edematous reactions at every site of previous application of a local anesthetic on administration of sulfanilamide by mouth.[14]

There is some evidence that the hypersensitivity reaction to sulfonamides may predispose the patient to further toxic effects on the central nervous system. It is certainly true that delirium and a toxic psychosis frequently accompany the febrile reaction. Little[2] has ascribed other neurologic manifestations, such as peripheral neuritis, to intermittent administration of the

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drugs. Longcope noted one case of coma and encephalitis following a hypersensitivity reaction.[15]

BLOOD DISORDERS

Blood disorders such as agranulocytosis, acute hemolytic anemia and thrombocytopenic purpura are uncommon, but exceedingly serious complications of sulfonamide therapy. They are seen less often with sulfathiazole and sulfadiazine than with sulfapyridine or sulfanilamide therapy. It is not clear whether these are the result of hypersensitivity or whether they may not be due, in some cases, to the direct toxic effect of the sulfonamides.

Acute hemolytic anemia, though rare, occurs early in the course of sulfonamide therapy and is usually seen during the third to fifth day of treatment.[16, 17] It is characterized by a rapid decrease in erythrocytes, jaundice, urobilinuria, leukocytosis, reticulocytosis and, at times in severe cases, hemoglobinuria and uremia. It seems that these reactions are an expression of an individual idiosyncrasy to the drug, which, in some cases, may have been acquired through exposure to other compounds of a similar nature and, in other cases, may have developed during the sulfonamide therapy. This is suggested by the fact that readministration of the drug is often followed by a similar episode.

A more slowly developing anemia associated with hemolysis of erythrocytes is not infrequently seen with sulfanilamide and especially with sulfapyridine therapy, but is rarely seen in sulfathiazole or sulfadiazine therapy. It is usually not an important complication of therapy since it is easily controlled by transfusions.

Thrombocytopenic purpura has been noted after sulfathiazole and sulfadiazine administration, as well as with administration of the earlier sulfonamide compounds. Too few cases have been studied to be certain whether it is the result of a direct toxic effect on the bone marrow or an individual idiosyncrasy to the drug. In some of the cases reported, the thrombocytopenia developed early in the course of sulfonamide therapy, on the second or third day, which might suggest a direct toxic effect or an idiosyncrasy developed by previous exposure to related compounds. In other cases, the purpura was seen after six or more days of therapy or on readministration of the drug. It is possible that, in these cases, a sensitivity to the drug developed during the course of therapy. Readministration of the drug following recovery from such a reaction may promptly precipitate another attack.[18, 19] Gorham *et al.*,[20] on the other hand, call attention to the similarity between the clinical signs of the hematotoxic action of benzene and aniline and those of the chemically related sulfonamide compounds. Kracke[21] has shown that the platelet count tends to decrease on the first day of sulfathiazole therapy, with a decided increase in the number of platelets on the first day after the drug is discontinued. This might suggest that the sulfonamides exert a primary toxic effect on the platelets.

In contrast to acute hemolytic anemia and thrombocytopenia, it is uncommon for leukopenia or agranulocytosis to occur early in a course of sulfonamide therapy. The decrease in the white blood cells is not usually encountered before the end of the second week and is more frequently seen between the fifteenth and twenty-fifth days of therapy. This reaction is seen especially with the use of sulfapyridine, but is occasionally a complication of sulfathiazole and sulfadiazine therapy.[22] In some cases, it appears to be the result of an idiosyncrasy that has developed during treatment, since readministration of small amounts of the drug will cause a rapid fall in white cells, including granulocytes. In other cases, readministration is not attended by changes in the blood picture.[1] This is a particularly important point since it has been generally assumed that the association of agranulocytosis with sulfonamide therapy was the result of an idiosyncrasy. Since sulfonamides would be especially important in controlling the serious secondary infection associated with agranulocytosis, it is possible that they should be continued in spite of the agranulocytosis. Four patients with agranulocytosis that developed after sulfadiazine therapy recovered completely with the prompt readministration of the drug, and spontaneous regeneration of the granulocytes occurred during sulfadiazine therapy.[23]

Another type of leukopenia and agranulocytosis has been described in rats and may have its clinical counterpart in man when sulfonamides are administered over a considerable period of time. It was found that the administration of succinylsulfathiazole or sulfaguanidine to rats fed a purified diet produced a leukopenia and agranulocytosis which would respond to the administration of liver or liver extract.[24,25] It is suggested that the sulfonamide compounds may act to produce this picture through a lowering of the intestinal synthesis of essential growth factors. It is possible that some of the cases of agranulocytosis noted in man after prolonged administration of sulfonamides may be the result of a disturbance in folic acid metabolism and the subsequent failure of maturation of white blood cells, rather than a direct toxic or sensitivity effect. Under such conditions, the favorable response to liver extract therapy may be expected.

URINARY PRECIPITATION

A not uncommon and usually avoidable complication of sulfonamide therapy is the precipitation of the compounds or their acetyl forms in the kidney tubules or ureters in sufficient amounts to cause mechanical obstruction to the flow of urine. Since these drugs and their acetyl forms are weak acids that are relatively insoluble in water or the normally acid urine, precipitation is very likely to occur as the alkaline ions and water are reabsorbed from the glomerular filtrate in the tubules of the kidney, producing relatively high concentrations of the drug in an acid environment. The amount of precipitation and the complications resulting from it vary con-

siderably with each of the compounds now in use, depending on the differences in the solubility of the free and acetyl forms and on the rate of excretion of the drug. Sulfanilamide, the most soluble of all the compounds, is rarely associated with clinically significant precipitation. Sulfathiazole, which is excreted very rapidly, has only a small proportion in the acetyl form, but, because of the rapid rate of excretion, high concentrations are very likely to occur in the urine, with subsequent precipitation. On the other hand, sulfapyridine and sulfadiazine are excreted more slowly and will consequently have a greater proportion of the drug in the more insoluble acetyl form; which favors precipitation. It is because of this that compounds, such as sulfamerazine and sulfamethazine, have been developed which are slowly excreted and have an acetyl form that is as nearly soluble or more readily soluble than the compound itself.

It is not uncommon to find crystalluria in patients receiving sulfonamides, and in itself this is of little significance, since crystals are likely to form as the urine cools from body temperature. On the other hand, crystalluria in a warm freshly voided specimen is evidence that precipitation is occurring in the urinary tract, although symptoms may not be present. At times, renal or ureteral pain, hematuria, burning on urination or oliguria are the first indications of significant precipitation of crystals. This may go on to oliguria with renal retention, of not only nitrogenous products, but also the sulfonamides, causing acetylation to become more extensive and thus promoting a tendency to further precipitation and complete repression of urine.

Fortunately, these complications can usually be avoided. This is due to the fact that the solubility of these drugs and their acetyl forms may be greatly enhanced in an alkaline medium so that they may form the soluble sodium salts. Thus, a change in pH of the urine from 6.5 to 7.5 may increase the solubility of sulfadiazine ten times.[26] The solubility of free or acetyl sulfathiazole and sulfadiazine greatly increases as the pH of the solution is raised above 7, so that it is of great importance to maintain an alkaline urine while these drugs are administered. The maintenance of a large volume of urine may also aid in the removal of the compounds without precipitation, but is of relatively slight importance in comparison to the alkalinization of the urine.

Though the incidence of untoward complications with the sulfonamides is not high, especially with the use of sulfadiazine, deaths directly attributable to the drugs do occur. Sutliff *et al.*[27] estimated that, in New York City, there was one death in every 1,610 cases of pneumonia resulting from complications associated with sulfonamide therapy. Long[1] has estimated that 5,000 deaths a year result from the use of sulfonamides.

Fortunately, many deaths and severe sulfonamide reactions may be prevented by intelligent use of these drugs. Careful consideration must be given to the reasons for instituting sulfonamide therapy in a particular case,

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and the drugs should be avoided when there is only a remote possibility that such therapy may have a beneficial effect. Care should be taken also to determine, as nearly as possible, the patient's previous experience with the drugs and the type of compounds previously administered.

Adequate supervision of the patient receiving the drugs will often prevent serious complications. This does not necessarily imply extensive laboratory procedures, but does involve careful clinical observation and occasional laboratory tests. During the first few days of administration of the drug, the patient should be observed for toxic effects on the central nervous system. In patients who have received a course of sulfonamides in the past, it is especially important to be alert during the first few hours of the readministration of the drug for the possibility of a hypersensitivity reaction, especially when large doses are administered. During the first few days of treatment, the rare complications of an acute hemolytic anemia or thrombocytopenic purpura might be anticipated. The urine should be examined for evidence of a direct nephrotoxic action of the drug and also to evaluate the presence of crystalluria.

Between the second and third weeks of therapy, the hypersensitivity reaction is most likely to develop, as well as the serious complication of agranulocytosis. White blood cell counts at this time are particularly indicated. Careful clinical observation of the patient may abort a hypersensitivity reaction before it becomes serious. The sudden development of malaise, arthralgia, conjunctivitis, skin rash or fever without other apparent cause should suggest such a reaction, and immediate cessation of therapy may abort the attack. Beyond the third week of therapy, the possibility of the development of an agranulocytosis must always be kept in mind. At all times during sulfonamide therapy, it should be remembered that the drug is being removed by the kidneys and that, in an acid urine, it is relatively insoluble so that crystals are apt to form. Since the solubility of the drug is greatly enhanced by alkalinity of the urine, the simultaneous administration of sodium bicarbonate is beneficial. The common practice of administering sodium bicarbonate in equal amounts with the sulfonamide will not necessarily insure an alkaline urine, and it is often necessary to administer larger amounts of alkali. Frequent examination of the urine to be certain that it has an alkaline reaction, as well as a careful maintenance of an adequate urinary output, will be helpful in preventing mechanical obstruction of the urinary tract.

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UMI

MANAGEMENT OF EARLY ACUTE DENTO-ALVEOLAR INFECTIONS

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EARLY acute infection about the jaws has had considerable ill-management. Perhaps the fact that, in many instances, surgical intervention in the presence of acute inflammatory conditions has had a favorable outcome has served to establish in the minds of many the idea that surgical procedures can be carried out with little regard for acute infection; that a resultant fulminating infection is destined to occur, and that surgical interference would not be a contributing factor. Disproof of this idea must lie in the records of the thousands of cases handled conservatively as against those of cases undergoing inopportune radical procedures. One should need only to see a case of cellulitis of the face and neck that has followed the mere incision of an acutely inflamed mandibular third molar gum flap, or see an exacerbation of soft tissue infection follow tooth extraction, to recognize the possibility that untimely surgical procedures may invite disaster.

This writing is intended to set forth, more for the attention of the dentist in general practice who practices minor oral surgery only occasionally, some of the concepts of good practice relative to this subject. These principles have been stated here and there in the literature, but it is my opinion that they merit assemblage and repetition as a single topic with a view to presentation in their specific relationship to the various types of infection problems that confront the dentist.

Decision as to the management of an acute infection becomes a matter of judgment based on a knowledge of the processes of inflammation, and to that end a résumé of those processes is here given.

Inflammation, a tissue reaction to irritation or injury, is, within the limits of this presentation, the defense reaction to invading bacteria and their toxins.

The weapons with which Nature has endowed the body to resist infection fall into two classes, the chemical and the physical.[1] The chemical substances consist of antibodies and antitoxins, which appear in the blood serum and lymph; while the physical substances are the phagocytic cells such as polymorphonuclear leukocytes and lymphocytes and, to some extent, connective tissue.

When pathogenic micro-organisms gain admission to the body tissues, large amounts of the antibodies are formed in the circulating blood to combat the invading bacteria. These antibodies are specific for the micro-organisms that have brought about their production. Antitoxins, formed

at the time of bacterial invasion, have the parallel function of neutralizing the toxins which would otherwise injure tissues.

The physical substances, primarily the white blood cells, although formed remotely in the body, congregate and act at the site of inflammation to control the local phenomenon. In the case of the acutely infected pericoronal tissues of a partially erupted mandibular third molar, for example, severe local disturbances may arise. The initial phase is usually marked by tenderness of the overlying gingival tissue, and occurring simultaneously are the signs of swelling and redness of the flap. Here, the bacteria have gained entry through the mucous membrane and Nature is already fighting their multiplication and spread into the deeper tissues. Leukocytes escaping from the blood stream directly into the tissues of the infected region concentrate in a peripheral zone around the region of infection, and, by phagocytic action on the bacteria, serve as a barrier to the further passage of micro-organisms into the body. Concurrently, the inflammatory exudate, or altered blood serum, escapes from the vessels into the affected tissue and edema results. Usually, there is evidence of regional lymph gland swelling and tenderness, indicating that the bacteria are being filtered out as they disperse along the lymph channels. This lymphadenitis is considered an aid in determining the stage of spread of an infection and can be taken as a sign of incompletely controlled bacterial activity. Lymphadenitis may, in general, be considered as warning against immediate surgical intervention in early infections.

We may think of the tissues involved in an inflammatory process as passing through a crisis in the battle between the invading bacteria and the resistance mechanisms of the body. As readily in the early phases of acute infection as in any other, the bacteria may gain ascendancy and the infection may spread dangerously out of control. The chief factors in their spread and multiplication are: (1) the low resistance of the patient; (2) the high virulence of the micro-organisms, and (3) the showering of bacteria into the neighboring region by unwarranted manipulation of acutely inflamed tissue. It is particularly the last factor that often seems to be insufficiently understood, and it is the purpose of this discussion to give thought to those situations which call for special consideration. Those conditions are: (1) acute pericoronal infection; (2) acute pericemental infection, or acute infection of pyorrheic pockets; (3) acute apical infections, and (4) surgical procedures in the presence of Vincent's infection.

In a preceding paragraph, the bacteriologic picture of acute pericoronal infection was used as an example. Treatment of the earliest stages of infection, when there are tenderness, congestion and swelling of the flap, is important for prevention of untoward sequelae. Errors in treatment may seriously aggravate the condition, producing within a short period a phlegmon of the face and neck. (The especial danger of infections

originating in the mandibular third molar region lies in the fact that the fascial plane drainage is into the masticator space[2] rather than into the more self-limiting mandibular space with which the other mandibular teeth are associated.) Not infrequently, it has been observed that stretching of an acutely infected gum flap during treatment has served as sufficient trauma to intensify symptoms.

The one procedure most to be censured is the incision of an acutely inflamed pericoronal flap for the purpose of exposing the gum pocket and relaxing the tissue. Incision or any unnecessary manipulation of acutely inflamed tissue is apt to break down Nature's wall of resistance in and about the region, permitting microbes to enter unfortified surrounding tissue where they may multiply and spread unchecked. It is true that certain oral surgeons favor incision of the overhanging ledge of gingival tissue to lay open the pocket, but if this is done at all, it is not to be done in the acute inflammatory stage. It is my conviction that conservative treatment is the most favorable, and the following procedures are suggested on this basis.

Where overlying gum tissue lies tight against the crown of the third molar, instillation of a suitable germicidal agent, e.g. saturated copper sulfate solution or iodine lotion, by means of a blunt cannula beneath the flap, can be carried out without injury to the tissue. Where the flap is ample and loose, or where a gingival crevice is accessible, the application of a germicide on a wisp of cotton, allowing a few minutes for penetration, will usually be found satisfactory. Often, a single treatment is adequate. If upper third molar cusps are traumatizing swollen lower retromolar soft tissue, the upper tooth may advantageously be extracted immediately, provided Vincent's infection is not a complicating factor. Removal of the mandibular tooth is not advisable, however, until the edema of the pericoronal tissues and the accompanying lymph gland tenderness have subsided and have remained negative for several days.

The acute pericemental abscess affords an opportunity for a dentist to follow a conservative course and avoid complications. In this condition, when a mucoperiosteal abscess is forming, the infection is usually fairly well established before the patient is seen. Starting usually from a pyorrheic pocket, the abscess tends to progress rapidly by extending apically and laterally beneath the submucosa. In these cases, there is a tendency for the process of infection to be self-limiting because of early localization. Spontaneous rupture of the abscess is common, and the period of discomfort is apt to be short. Nevertheless, the dentist is frequently prevailed on to extract in the early stage because of the attendant symptoms. If extraction is performed during the period of mucoperiosteal swelling, the danger lies not only in affecting adversely the course of the soft tissue infection, but also and particularly in the risk of direct con-

tamination of the alveolus. Under this circumstance, there is the immediate possibility of a resultant osteomyelitis.

Proper early treatment should consist of waiting for satisfactory localization of pus to permit incision and drainage. Frequently, drainage can be effected by gently probing down into the pyorrhea pocket and along the periosteum in the direction of the abscess. The use of intra-oral heat in the form of hot saline irrigation or rinses is an acceptable procedure, but the use of extra-oral heat in this or any other early acute dento-alveolar infection is contraindicated. Whether incision and drainage are carried out or the infection subsides by resolution without suppuration, extraction should not be anticipated for several days, at least until the swelling and tenderness have subsided.

Acute apical abscesses call for careful evaluation of signs and symptoms for selection of the best course of treatment. Inflammation of bone is essentially the same process as in soft tissue, except that bone is entirely unyielding, and any engorgement of the blood vessels or extravasation of lymph therefrom is manifest by deep, boring pain in the jaw. The use of the roentgenogram is important for verifying the clinical diagnosis and for determining the nature and extent of periapical bone rarefaction.

It is difficult to give inflexible rules regarding indications or contraindications for extraction in all cases of acute apical infection. However, infections involving the upper anterior teeth are in a category by themselves, and one important rule may be given: do not extract an upper incisor or cuspid tooth in the presence of an acute infection. If the presence of pus is suspected, and it appears to be confined within the bone, some drainage and release of pressure may be established by way of the root canal. But it is important in this instance that care be taken not to disturb the periapical cancellous bone lest the infection spread farther. Sulfonamide therapy may be especially valuable from the outset in these cases of infection in the premaxillary region.

As to the mandibular teeth or the maxillary posterior teeth, it can be said that, in general, early extraction in acute apical infections is advised if there is clinical evidence that the inflammation is confined to bone. Extraction should be performed with the least amount of trauma possible. Disturbance of the socket, as by curettage, is contraindicated. When there is evidence that the abscess has perforated the cortical bone and when soft tissue involvement is noted, extraction is contraindicated. Serious exacerbation of infection has frequently resulted from tooth extraction in the presence of such a seemingly innocuous sign as moderate tenderness over the apical region. It naturally follows that a patient presenting himself for extraction of a tooth with an acute periapical abscess rightfully should be apprised of the dangers involved if extraction is carried out or if it is not.

When the stage of mucoperiosteal swelling or regional edema has been

reached, incision and drainage should be anticipated, and they should be carried out when the abscess is localized and fluctuant. The abscess is usually found to be localized in the region of the mucobuccal fold. As often happens, a soft tissue abscess does not materialize, but subsides by resolution without benefit of surgical measures. This is a point to be kept in mind when localization of pus is uncertain. Frequently, this is the case in the indolent swellings occurring in infections with micro-organisms of low pathogenicity.

The oral and parenteral administration of sulfonamides[3] in acute dento-alveolar infection holds an important place in the oral surgeon's management of these conditions, but their routine use in all early infections is not yet accepted practice. In the first place, their use should be reserved for the more serious situations; secondly, sole responsibility for the administration of these drugs should not be assumed by anyone not entirely familiar with their action. It would be difficult to attempt to draw a definite line of distinction between those situations wherein the use of the drug would be considered necessary and those in which it is not called for. As mentioned previously, they may be justifiably used in early involvement of upper anterior teeth because of the unusual hazard of infection in that region. Sulfathiazole or sulfadiazine could be prescribed when there is marked submaxillary lymphadenitis, such as is seen with severe pericoronal infections. Otherwise, for conditions less grave, for those early, uncomplicated dento-alveolar infections, sulfonamide therapy is not generally considered indicated.

The use of extra-oral heat in the early stages of any of the acute dento-alveolar infections is generally inadvisable because of the predisposition to cellulitis. When slight soft tissue swelling already exists, the use of extra-oral heat can be expected to increase the degree of swelling and accompanying symptoms by guiding the process of infection in the direction of the applied heat. Usually, the less edema, the more favorable the general situation. On the other hand, the extra-oral application of cold is a more rational procedure, but its use does not often appear to be indicated and, in some instances, may be detrimental. In the instance of a moderate lymphadenitis, the conservative application of cold over the affected part may be good practice. When there is slight soft tissue edema over the region of a subperiosteal abscess, the application of cold may have no particular value and may even retard the natural process of inflammation and repair. In general, the early, simple edema attending infection may be more favorably influenced without the use of extra-oral heat or cold.

The dangers of executing surgical procedures in the presence of acute Vincent's infection cannot be overemphasized. By and large, the disease is immediately recognized and suitable treatment is given to bring it under control before any surgical step is undertaken. However, bad cases of

infection are occasionally seen in which an extraction has been carried out, a third molar gum flap incised or a radical prophylaxis performed.[4]

It has been said by others many times before, and it can be said once more, that thorough examination should be made when there is evidence of Vincent's infection, and when the infection is present, it should be treated until eliminated before any surgical measurers are undertaken in the mouth.

CONCLUSIONS

It must be recognized that "inflammation is not a disease, but a manifestation of Nature's mechanism of combating infection, and as such should be respected."[1] Consequently, the conservative approach to the management of acute infections is in the end the safest procedure. For the most part, unpleasant as it may seem to the patient, "awaiting developments" can be expected to have a more fortunate outcome than irrational surgical interference in the presence of acutely inflamed tissues.

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Apicoectomy—Postoperative Observations.—Just what takes place in the repair of the wound after this operation is what interests us most. It has been our observation that the repair of bone after this operation does not differ from bone repair in any other part of the membranous bones. Neither does the repaired bone differ materially from the normal bone. In opening into these areas periodically from six months to several years after the operation, we find that the formation of new bone seems to be clinically identified with that adjacent to the old wound. From our bacteriologic findings, this new bone has remained sterile. We believe that if the principles above referred to are carried out, an operation may be performed that will favor normal repair. There are three requirements for bone repair all of which occur in these wounds: (1) blood supply; (2) building material; (3) stimulus, physiologic or pathologic. The rich blood supply in the maxillae and mandible amply provides for the first requirement in the regeneration of new bone. —Chalmers J. Lyons, in *J.A.D.A.*, 1928.

FURTHER STUDIES ON THE EFFECT OF THE PROPHYLACTIC USE OF SULFATHIAZOLE AND SULFAMERAZINE ON BACTEREMIA FOLLOWING EXTRACTION OF TEETH

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IN a previous report,[1] it was noted that the prophylactic use of sulfathiazole greatly reduced the incidence of bacteremia following the extraction of teeth. It was shown that the drug must be given in sufficient quantity to raise the blood level at the time of operation above 4.0 mg. per hundred cubic centimeters. This investigation has been continued in order to observe a large series of cases. At the same time, a series of cases using sulfamerazine as the prophylactic agent have been studied.

In order to evaluate these findings, certain basic information discussed in some detail in the original report will be presented here.

It is now well established by both medical and dental research that, after the trauma from manipulation or surgical procedures, showers of organisms are carried into the blood stream. The incidence of bacteremia of this type varies, depending on the type of anesthesia used. When extraction is performed under general anesthesia, bacteremia has been shown to occur in about 64 per cent of the cases reported,[2] when only one or two teeth were removed. When teeth are extracted under local anesthesia, the incidence has been about 17 per cent.[3, 4] This wide variation in the incidence of bacteremia with the two types of anesthesia can be attributed to the vasoconstrictor action of the adrenalin incorporated in most local anesthetics.

SIGNIFICANCE OF TRANSIENT BACTEREMIA

While, in the normal person, bacteremia has little significance, in the person who has preexisting valvular heart disease, bacteremia may precipitate a subacute bacterial endocarditis.

CONTROL SERIES

It seemed necessary to establish a series of control cases before studying the effect of sulfathiazole and later sulfamerazine therapy on transient bacteremia following the extraction of teeth. Thus, it would be possible to check previous reports and also to standardize our own procedure. No attempt was made to select any specific type of case or even to limit the number of extractions to one or two teeth. All types of extractions were

included in the series. The surgical procedures were carried out by students, using 2 per cent procaine with 1 : 50,000 adrenalin. The bacteriologic technics will not be given in this article, but can be obtained by referring to the original report.[1]

Cultures taken immediately on completion of the operation, as seen in Table 1, showed an incidence of 16.1 per cent positive, which compares favorably with the 17 per cent usually reported by other workers. (The percentage of positive cultures reported here is slightly greater than in the last report.) It might be reiterated that cultures taken before operative procedure and ten minutes after the completion of the operation were negative with two exceptions. The exceptions were among the cultures preliminary to the operation. The organism isolated in both of these cases was *Staphylococcus albus,* and we considered its presence to be the result of contamination.

TABLE 1.—CONTROL GROUP—NO THERAPY: RESULTS OF CULTURES IMMEDIATELY FOLLOWING OPERATIVE PROCEDURES

Bacteriologic Findings	No. Times Isolated
Contamination	3
Streptococcus viridans	7
Streptococcus viridans and Staphylococcus albus	1
Streptococcus viridans and actinomyces	1
Anhemolytic streptococcus	3
Anhemolytic streptococcus and actinomyces	1
Anaerobic streptococcus	1
Actinomyces	2
Number of positive cultures	16=16.1%
Number of negative cultures	83=83.9%

DISCUSSION

From our study, it appeared that the prophylactic use of sulfathiazole, properly administered, decreased the incidence of bacteremia following the extraction of teeth. To obtain the most favorable results, certain important conclusions relative to administration of the drug need further emphasis and clarification, if one wishes to apply them to technical procedures. Table 2 presents the results obtained by the routine method of premedication with sulfathiazole. This method consisted of giving 1 gm. of the drug and a like amount of sodium bicarbonate every four hours until 6 gm. of sulfathiazole had been taken by the patient, the last dose having been taken three hours before the operation was performed. In this series, two groups are presented. Group A consisted of forty cases in which blood levels of the drug were found to be over 4.0 mg. per hundred cubic centimeters. Group B were cases in which the blood level was below 4.0 mg.

In Group A, the incidence was found to be 5 per cent of positive blood cultures with an average blood level of 4.6 mg. per hundred cubic centimeters. Group B comprised a series of fifty-five cases which show an incidence of 14.5 per cent of positive blood cultures with an average blood level of 2.9 mg. per hundred cubic centimeters. These findings revealed the importance of raising the blood level of the drug above 4.0 mg. if the optimum results are to be obtained. It also shows the difficulty of attaining such a result by this method of drug administration since 58 per cent of the ninety-five cases showed a level below 4.0 mg. per hundred cubic centimeters. This variation of the blood level is probably due to the individual ability of patients to absorb and excrete the drug, and to failure on

TABLE 2.—SULFATHIAZOLE ROUTINE GROUP: RESULTS OF BLOOD CULTURES IMMEDIATELY FOLLOWING OPERATIVE PROCEDURES

Bacteriologic Findings	No. Times Isolated	Sulfathiazole in Blood Mg.*
Group A: Blood Levels Above 4.0 Mg.*		
Contamination	3	4.4; 5.1; 5.3
Anhemolytic streptococcus	1	5.3
Streptococcus viridans	1	5.2
Total number of positive cultures	2=5%	
Total number of negative cultures	38=95%	Ranged from 4 to 6.5 Average=4.6
Group B: Blood Levels Below 4.0 Mg.*		
Contamination	2	3.0; 3.3
Streptococcus viridans	7	2.9; 2.8; 2.3; 3.0; 0.4; 1.2; 3.5; 3.6
Actinomyces	1	0.1
Total number of positive cultures	8=14.5%	
Total number of negative cultures	47=85.5%	Ranged from 1.6 to 3.9 Average=2.9

*Per hundred cubic centimeters.

the part of the ambulatory patients treated to cooperate by taking the drug every four hours, especially during the night.

Because of the disadvantages of this method, a series of cultures was taken from patients who had been given one massive dose of 5 gm. each of both sulfathiazole and sodium bicarbonate three hours before dental extractions. The results are shown in Table 3. Thirty-one of the total of forty-four cases showed blood levels over 4.0 mg. per hundred cubic centimeters. The average was 5.8 mg. An incidence of 3.2 per cent of positive cultures was found in this group. Group B consisted of thirteen patients in whom the blood level was found to be below 4.0 mg., with an average of 3.4 mg. The incidence of positive cultures in this group was 0.8 per cent, which, con-

sidering the size of the group, has little significance. From this study, it appears that one massive dose is a more effective means of raising the blood to the desired level than the original procedure. Even the average blood levels in the group below 4.0 mg., when the massive dose was used, were found to be considerably higher than in the corresponding group following the usual method.

Table 4 is a composite of the two groups treated by sulfathiazole (Tables 2 and 3). Group A shows the results of seventy-one cases in which the blood level was above 4.0 mg. per hundred cubic centimeters. An incidence of 4.2 per cent of positive cultures was obtained. Group B consists of fifty-eight patients whose blood levels were below 4.0 mg., with an incidence of 14 per cent of positive cultures.

TABLE 3.—SULFATHIAZOLE SPECIAL THERAPY: RESULTS OF BLOOD CULTURES IMMEDIATELY FOLLOWING OPERATIVE PROCEDURES

Bacteriologic Findings	No. Times Isolated	Sulfathiazole in Blood Mg.*
Group A: Blood Levels Above 4.0 Mg.*		
Contamination	1	4.7
Streptococcus viridans	1	8.6
Total number of positive cultures	1=3.2%	
Total number of negative cultures	30=96.8%	Ranged from 4.1 to 9.2 Average=5.7
Group B: Blood Levels Under 4.0 Mg.*		
Staphylococcus aureus	1	3.2
Total number of positive cultures	1=0.8%	
Total number of negative cultures	12=99.2%	Ranged from 2.5 to 3.9 Average=3.4

*Per hundred cubic centimeters.

Some time ago, we became interested in the possibilities of sulfamerazine as a bacteriostic agent, and its action on bacteremia following the extraction of teeth. Since this drug has been thought to be less toxic than sulfathiazole and to be absorbed rapidly and excreted slowly[5] (as recently reported by Hall and Spink), which would be a great advantage since a smaller amount of the drug would be necessary in order to reach and maintain the desired blood level, it would appear to have qualities superior to those of sulfathiazole.

Table 5 reveals the results of blood cultures taken immediately after operation when 2 gm. of sulfamerazine and sodium bicarbonate had been given three hours before operative procedures were carried out. Group A consists of thirty-nine cases in which the blood levels were above 4.0 mg.

per hundred cubic centimeters. In fact, an unusually high average of 8.2 mg. was obtained. In this series, six positive cultures, or an incidence of 15.3 per cent, were found. Group B comprised only six cases with a blood level below 4.0 mg. No positive cultures were obtained. This group again is too small to be of much significance. Group A is very significant, for it appears that sulfamerazine is not a good bacteriostatic agent in *Streptococcus viridans* infections. We believe that its use as a prophylactic in the management of patients with valvular heart disease is undesirable.

From this investigation, it seems that prophylactic premedication by sulfathiazole should be instituted in the patient having valvular heart disease, if the infection is present or surgical measures are contemplated.

Sulfanilamide, sulfapyridine and sulfadiazine may be as efficacious as or even more efficacious than sulfathiazole. However, at the moment, there is not sufficient scientific information on their merits for prophylaxis in valvular heart conditions. It is our desire to study in the same manner as we have investigated the prophylactic use of sulfathiazole the effects of their employment.

TABLE 4.—SULFATHIAZOLE TREATED GROUP*

Group A: Blood Levels Above 4.0 Mg.†	
Number of positive cultures	3=4.2%
Number of negative cultures	68=95.8%
Group B: Blood Levels Below 4.0 Mg.†	
Number of positive cultures	9=14%
Number of negative cultures	59=86%

*Combination of Tables 2 and 3.
†Per hundred cubic centimeters.

It should be emphasized that sulfathiazole should not be used promiscuously and that its use is advocated for patients whose histories show a predisposition to endocarditis. At the present time, the sulfonamides are being used indiscriminately for many minor ailments, such as upper respiratory infections, and sinusitis, and even before the extraction of carious teeth. Such use of these drugs is condemned, for not only is it of doubtful therapeutic value in these cases, but it is also extremely dangerous to the patient should an idiosyncrasy develop. Further, if such a reaction occurs, the patient cannot have the advantages of the drug later when a serious illness occurs.

Medical men whom we have consulted during the past four years have accepted our findings enthusiastically as a contribution to the management of the patient with endocarditis. Not only has the physician cooperated in the medication of these patients, but he also has the opinion that such patients should be given postmedication for at least twenty-four to thirty-six

hours, in order to maintain the drug in the blood stream at the proper level. Such treatment probably is the safest procedure.

Although, in this study, we have not encountered any drug reactions, it is suggested that a physician prescribe the drug, for, on rare occasions, a patient may show an idiosyncrasy to even a small dose or single dose. Should such a complication arise, the physician can not only share in the responsibility, but also is better able to treat the situation.

In the management of the endocarditis case given premedication, the question arises as to how much surgical work should be performed at one time. It is our opinion that once there is adequate premedication, as much surgical work as possible at one time should be completed. Of course, such

TABLE 5.—SULFAMERAZINE-TREATED GROUP: RESULTS OF BLOOD CULTURES IMMEDIATELY FOLLOWING OPERATIVE PROCEDURES

Group A: Blood Levels Above 4.0 Mg.*

Bacteriologic Findings	No. Times Isolated	Sulfamerazine in Blood Mg.*
Contamination	3	0.50; 5.5; 0.0
Streptococcus viridans	4	4.2; 5.0; 5.9; 8.6
Streptococcus viridans and actinomyces	1	8.3
Actinomyces	1	4.8
Total number of positive cultures	6=15.3%	
Total number of negative cultures	33=84.7%	Ranged from 4.0 to 10.0 Average=8.2

Group B: Blood Levels Below 4.0 Mg.*

	Sulfamerazine in Blood	
Positive	0	0
Negative	6	2.9; 2.0; 3.0; 3.6; 3.7; 3.8 Average=3.3

*Per hundred cubic centimeters.

factors as the general health of the individual, the difficulty and extent of the operation and the degree of the infection influence this decision. We should keep in mind constantly that a sulfonamide should be used the least possible number of times.

A word might be added here about the importance of eliminating all questionable foci of infection in this type of patient. Subacute bacterial endocarditis may follow a severe upper respiratory infection; in fact, any acute infection or injury. Therefore, the physician and dentist should eliminate any questionable foci so that an acute complication will not arise later and thus predispose this patient to subacute bacterial endocarditis.

CONCLUSION

As was stated in the previous publication, it is the responsibility of the

dentist and physician to afford patients who have valvular heart disease added safety by the prophylactic use of the sulfonamides. The dentist should inquire routinely into the patient's past history to determine whether the patient has ever had rheumatic fever, chorea or any heart disturbance. This inquiry should be made of patients with severe oral sepsis or when surgical procedures are contemplated. If, from this inquiry, possible or definite information suggesting valvular heart disease is obtained, the dentist should contact the patient's physician requesting his confirmation or diagnosis. If his examination suggests the mere possibility of valvular heart disease, sulfonamide premedication should be prescribed. The physician likewise has a responsibility in the management of the patient with congenital heart disease or endocarditis. Once a physician has under his care a case presenting questionable or recognized valvular heart disease, the patient should be considered definitely in the need of prophylactic treatment when any type of surgical operation becomes necessary.

SUMMARY

The prophylactic use of sulfathiazole, if given in doses sufficient to raise the blood levels above 4.0 mg. per hundred cubic centimeters, reduces the incidence of positive blood cultures from 16.1 per cent (our control group) to 4.2 per cent.

It was found that administration of one dose of 5 gm. of sulfathiazole given three hours before operation affords the most effective premedication.

When sulfathiazole is given in doses insufficient to raise the blood level above 4.0 mg. per hundred cubic centimeters, the incidence of positive blood cultures is 14.0 per cent. This is almost the same percentage of positive cultures as in the control group (16.1 per cent).

Sulfamerazine therapy, while raising the blood levels sufficiently high as regards the amount of the drug present in the blood stream, does not prevent bacteremia following surgical procedures. This result is shown by an incidence of 15.3 per cent positive cultures, although the average blood level of thirty-nine patients was 8.2 mg. per hundred cubic centimeters of sulfamerazine.

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BLOOD AND LYMPH VESSEL TUMORS INVOLVING THE MOUTH

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EWING[1] defines these lesions as true neoplastic processes of both blood and lymph vessels, which thereby give rise to two classes of tumors, hemangioma and lymphangioma. Both apparently have their origin in sequestrations of blood or lymph vessel tissue and, in addition to being similar in origin, grow in an identical fashion by projecting buds of endothelial tissue. Likewise, the growth of both is subject to the element of mechanical pressure of the circulation, an influence that is absent in other tumors.

In support of the theory that these tumors are congenital is the fact that the large majority of them are observed at birth. The remainder are usually observed during the first few months of life, and undoubtedly many were present at birth, but remained undetected because of their minute size, or hidden location, or because of lack of a detailed oral examination. Also, they are often associated with other congenital malformations and anomalies such as spina bifida, hydrocephalus and cleft lip and palate.

DIFFERENTIAL DIAGNOSIS

Angiomas must be differentiated from other processes wherein there is an abnormal development of blood vessels. Pyogenic granulation tissue presents an excessive growth of blood vessels, seen protruding from a chronically infected tooth socket, the orifice of a suppurating fistula (Fig. 1) or a maxillary oral opening. These pathologic conditions are the result of subacute or chronic inflammatory changes, associated with various protective and reparative processes. They are self-limiting and promptly regress on removal of the irritant. Histologically, the blood vessels lack neoplastic characteristics.

Tumors of pregnancy (Fig. 2) are associated with stomatitis gravidarum, which occurs in some pregnant women about the second month of gestation. The accepted view is that their growth is due to stimulation incurred through hormonal activity associated with pregnancy. The tumors, which are deep red or blue, are attached to the gingival tissue by a pedicle. They are made up of angiomatous proliferation tissue and epithelial hypertrophied tissue. They regress after parturition, but treatment may be necessary before delivery because of their hemorrhagic tendency. In contradistinction to the condition in angiomas, the blood vessel hypertrophy of these tumors is self-limiting.

Likewise, angiomas are not to be confused with blood vessels in which

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chronic varicosities are established, as such blood vessels present a normal or nearly normal structure and do not exhibit neoplastic growth.

HEMANGIOMA

Hemangiomas may be single or multiple, unilateral or bilateral, and symmetrical arrangements occur. The skin, mucous membrane and internal organs may be the site of origin. While the head and neck make up less than one-seventh of the total body surface, more than one-half (56 per cent) of a series of 1,056 cases reported by Watson and McCarthy[2] occurred in this area. They may retain their original size, grow with the body or suddenly undergo rapid acceleration of growth. Ulceration or suppuration may occur from mechanical injury, an inflammatory process or infection. Life may be jeopardized by hemorrhage or sepsis.

These tumors make up the largest single group of neoplasms occurring

Fig. 1.—Pyogenic granulation tissue formation about orifice of suppurating oral-facial fistula. The vascular channels are of regular size, shape and distribution.

in childhood. Females are more frequently affected than males. They are rare in the Negro race.

There is considerable speculation as to the exact nature of the growth of these lesions. Some investigators believe that only a small percentage of them are true neoplasms, the remainder being considered hyperplastic blood vessels. The neoplastic type is so classified because of its ability to suddenly experience an accelerated, irregular and unrestrained growth. Through some process, buds of endothelial tissue grow into the adjacent normal tissue, where they become canalized and establish communication with the parent vessel. The newly formed vessel or space does not anastomose with the surrounding normal vessels, developing at the expense of

these vessels as well as all the surrounding normal tissue elements. The lesion possesses only one afferent and one efferent vessel.

CLASSIFICATION

Not all types of hemangioma are observed in the mouth, some forms being dependent on the skin elements for their development. The capillary and cavernous types are those most frequently seen in and about the mouth and face.

The capillary type, frequently referred to as angioma simplex or simple nevus, is most frequently found on the face and neck. It may vary in size from the head of a pin to involvement of a large cutaneous area. (Fig. 3.) It may vary from a bright red to a bluish red or wine red, the color depending on its structure and depth in the skin. Mechanical and thermal

Fig. 2.—Tumor of pregnancy. These growths are of varied histologic composition, but, in the main, are made up of fibrous tissue with angiomatous proliferation.

stimuli may influence its color temporarily and it may fade completely upon pressure; or it may retain a bluish brown hue, due to pigment deposits.

Histologically, angioma simplex consists of a network of capillaries situated superficially in the skin. A proliferation of the endothelial layers is frequently observed, resulting in the formation of new capillaries, with resultant thickening of the lesion, causing it to simulate the cavernous type. Closely associated with the capillary type of angioma is the so-called stellate or spider-web hemangioma (naevus araneus). It may appear as solitary or multiple, pinhead size lesions, with a slightly elevated central vessel from

which smaller vessels radiate, forming a figure resembling the body and legs of a spider.

The cavernous type of hemangioma is distinguished from the capillary type by its widely dilated spaces and channels. This is the type commonly affecting oral mucous membranes. Its size may vary from that of a pea (Fig. 4) to involvement of large areas or all of a structure, such as the tongue. (Figs. 5 and 6.) The lesions are soft, elevated and generally poorly circumscribed. If superficial, they are dark red, while the deeper types assume a dark bluish tinge. Changes in the circulation affect both the size

Fig. 3.—Capillary hemangioma of face with cavernous hemangioma of right upper lip. Mixed types are frequent. The cavernous portion involved the entire thickness of the lip tissue.

and the color. Many of the tumors are distinctly erectile and pulsating. They are soft and easily compressed, blanching readily on pressure and promptly refilling with blood when pressure is discontinued.

Cavernous hemangiomas have a greater thickness than the capillary type and extend deeper into the subcutaneous and submucous tissues. In the mouth, a multiplicity of lesions is a common feature of the small, benign type. In contrast with these is the large, single, invasive type of lesion

that exhibits certain peculiar features of malignancy. In the mouth, there is constant danger of severe hemorrhage from trauma in cases of large areas of involvement.

Mixed capillary-cavernous types are common (Fig. 3) and mixed hemangioma and lymphangioma are occasionally seen in the tongue, where both participate in the production of angiomatous macroglossia. The lips, buccal mucous membranes and tongue are the favored sites for hemangioma in the mouth.

Histologically, the cavernous hemangioma presents a series of anastomosing vascular channels enclosed by thin connective tissue septa lined with endothelial cells, all of which are connected with one main afferent and one efferent vessel.[3] On section, the appearance may be roughly compared to that of a sponge. The condition of the mucous membrane over-

Fig. 4.—Small cavernous hemangiomas of right lower lip. The lesions, which were raised, were a bluish purple. Treatment consisted of surgical excision. A much larger lesion in the floor of the mouth was destroyed by injection.

lying the lesion depends on whether the angioma is superficial or deep. If superficial, the mucous membrane may be markedly altered by tumor invasion and pressure atrophy, while, in the deeper types, the mucous membrane may be normal and freely movable over the lesion.

TREATMENT OF HEMANGIOMA

Since the majority of angiomas occur about the head, neck and face, treatment planning should include minimum disfiguration. The radiosensitivity of these lesions varies in inverse proportion to the patient's age, and it has been reported[2] that the percentage of regressions is higher at 3 months

of age than it is at 6 months with the use of the x-rays and radium. It is therefore important that if irradiation is to be the therapy of choice, treatment be instituted as soon as the lesion is detected. This fact should be called to the attention of pediatricians and obstetricians, with a plea for early treatment. The transition that takes place in the endothelial cell with age making it less sensitive to irradiation is not well understood.

Repeated application of carbon dioxide snow is effective in the management of superficial capillary lesions, provided they are free from pigment deposits. The extreme temperature injures the endothelial cells, causing thrombosis and fibrosis. Since the temperature change of the tissue is limited to but a few millimeters, this method of treatment is indicated only for the very superficial type of lesion. Efforts at deeper penetration by longer application of the thermal excess will result in ulceration. The carbon dioxide snow is cut into blocks of suitable shape and size. The blocks

Fig. 5.—Aggressive cavernous hemangioma of tongue. It was present at birth and had developed to the point where it interfered with speech and produced malocclusion of the teeth.

are held in contact with the lesion for a period of from ten to twelve seconds. Bleaching of the lesion takes place without scarring. Treatment is repeated at intervals, allowing time for each reaction to subside.

For the deeper types of capillary lesions, various surgical procedures are employed. In short, they entail the surgical excision of the involved skin and its replacement with full thickness grafts or flaps from adjacent or remote areas. (Fig. 7.) Wherever possible, the use of multiple partial excisions gives the best cosmetic result.[4] In early and middle life, the skin is elastic, rapidly regaining normal relaxation and elasticity after being put on marked stretch or tension. In planning such an excision, the final scar

should be so placed as to be as inconspicuous as possible. Permanent distortion of facial features should be avoided.

Briefly stated, the method consists of repeated excision of the lesion and closing of the defect by undermining the affected and normal skin. Judgment must be exercised as to the amount of involved skin to be removed each time in order to avoid undue tension in approximating the incised edges.

In areas unsuited for multiple excision, such as over the nose, where there is little elasticity to the skin, and about the eyes, where tension would produce distortion of the lids, flaps and grafts are frequently a necessity. While their cosmetic behavior is quite unpredictable, the use of full skin thickness flaps or grafts affords the best results.

The cavernous hemangioma involving the oral mucous membrane is best treated by surgical excision provided it has not attained such propor-

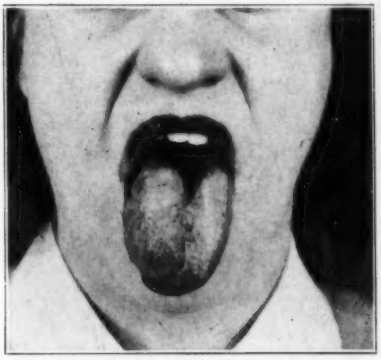

Fig. 6.—Result, eighteen months later, of sodium morrhuate injection at intervals of two months. (Patient shown in Figure 5.)

tions as to pass beyond the limits of justifiable surgery. However, many examples have been recorded in which a small lesion in an infant has become a formidable tumor in the adult. For these extensive lesions, especially those involving large portions of the tongue, the use of a sclerosing solution is the method of choice. Of the various sclerosing solutions employed, sodium morrhuate has uniformly given the best results. This treatment is based on the fact that hemangiomas are sensitive to irritation, many being known to regress spontaneously after slight injury. The drug action causes thrombosis of the blood channels with resulting fibrosis and elimination of the blood spaces.

Injection is effected with a blood needle of suitable length attached to a Luer syringe. A blood sinus is found and the blood aspirated. Compression of the afferent vessel will aid in preventing immediate refilling of the sinus

with blood. With the needle *in situ,* the syringe is detached and a second syringe containing from 1 to 5 cc. of the solution, the amount depending on the size of the lesion, is attached and the solution slowly injected. Pressure is applied as soon as the needle is withdrawn, being maintained until no blood leaks from the needle puncture. With repeated injection, it becomes more difficult to find individual blood sinuses. The solution is then injected diffusely through the lesion. In my experience, there have been no complications from emboli, nor does ulceration occur if the scleros-

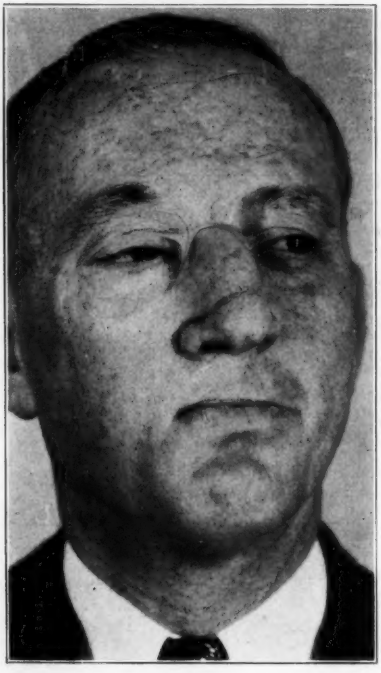

Fig. 7.—Patient shown in Figure 3. The cavernous portion of the lesion was excised. The involved skin of the cheek, temple and forehead was removed by multiple excisions. The upper and lower eyelids were corrected with full thickness grafts taken from the postauricular area and the involved skin of the nose was replaced with a pedicle flap from the forehead. The nasal flap and adhesion medially from the left eye have been corrected since the photograph was made.

ing solution is judiciously used. The local reaction is allowed to subside before another injection is made.

LYMPHANGIOMA

The clinical conditions included in the general class of lymphangioma

include a variety of slow-growing, single or multiple enlargements, usually present at birth, which, in the mouth, may be located in the lips, cheek or tongue. They have been observed in combination with other anomalies, especially hemangioma, and they have the same relationship to the lymphatics that a hemangioma bears to the blood vessels. It is generally agreed by most observers that they have their origin in displaced fetal cells of the lymphatic system. Like hemangioma, many possess marked growth capacity, but size is not always a reliable criterion of the neoplastic capabilities of the lesion.

A lymphangioma is regarded as a tumor composed of lymph vessels. Many different types of the growth are described as affecting different parts of the body, but, in the mouth, it is extremely rare to find other than the cavernous type. This form is composed of dilated lymphatic sinuses,

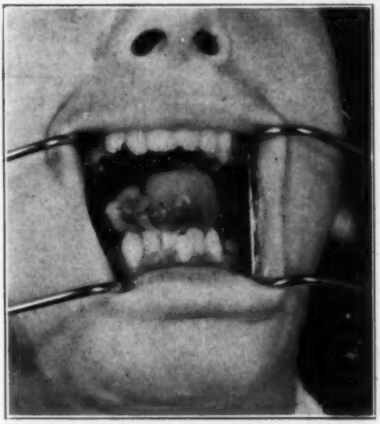

Fig. 8.—Mixed lymphangioma and hemangioma of tongue. The patient was unable to protrude the tongue beyond the teeth owing to fibrosis from repeated applications of radium.

usually filled with lymph. It is colorless unless it contains a number of additionally formed hemic structures, in which event it takes on a discoloration due to the presence of blood. (Fig. 8.)

In connection with the tongue, the affection may be limited to a definite area or it may involve its entire width and thickness, thereby producing marked macroglossia. (Fig. 9.) Curiously enough, this hypertrophy is limited to the anterior two-thirds of the tongue structure. As the growth increases, the tongue may become too large to be contained within the mouth and speech is interfered with. Despite the enlargement, pain is not present unless inflammation or ulceration occurs from injury. Deformity of the mandible may result.

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Histologically, the cavernous lymphangioma consists of a system of communicating lymph spaces lined with endothelium and filled with lymph that may be mixed with blood. As with hemangioma, solid endothelial buds can frequently be found in the adjacent tissue.

TREATMENT OF LYMPHANGIOMA

Many forms of treatment have been recommended in the past, but the employment of any therapy other than surgical is usually a waste of time and a source of considerable discomfort to the patient. In the case of the small circumscribed tumor, excision is complete, whereas the large diffuse types can be handled by a series of excisions. This is especially true of the tongue, where wedge-shaped excisions reduce the lesions to such a size that it can be contained within the mouth. Sclerosing solutions are ineffectual

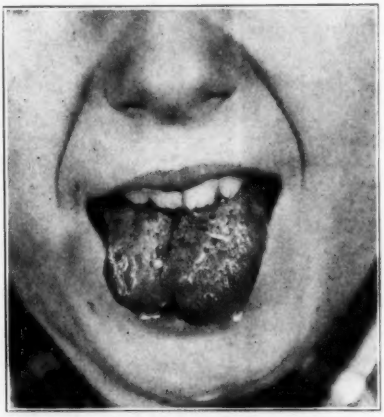

Fig. 9.—Lymphangioma of tongue. Wedge-shaped excisions from the anterior portion of the tongue control the macroglossia. Function is well preserved.

and lymphangiomas are radioresistant. The tendency to recurrence is rather strong and increases with age.

SUMMARY

1. Angiomas are tumors composed of newly formed blood and lymph vessels having their anlage in embryonic sequestration of mesodermal tissue.

2. The use of sodium morrhuate as a sclerosing solution for the larger intra-oral cavernous hemangiomas is advised.

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Oral Surgery in the Curriculum.—Previous to the beginning of the last quarter of the century of which we are reporting, oral surgery was taught in the dental schools in a very indifferent manner. The chair of this department was usually held by a physician or general surgeon with little or no experience in oral surgery, and instruction was given largely by didactic courses. Today, in practically all of the Class A dental schools, the course in oral surgery has become one of the most important in the curriculum. As dental education has advanced, and as a greater knowledge of the relationship of mouth infection to general systemic disease has been obtained, it has been found necessary for the dental student to have some hospital experience. To this end, all of the better dental schools have definite hospital connections through the department of oral surgery whereby the dental student comes in contact with hospitalized patients. This connection has done much to bring about a closer cooperation between medicine and dentistry. Today, practically all of the larger hospitals in America are requiring as a part of their staff dental interns who work on the same basis as medical interns. The dental intern mingles with the medical interns, speaks the same language and becomes the dental diagnostician of the hospital. A greater sympathy is thus created between the younger men of the two professions. Today, the large gap which existed between medicine and dentistry is gradually closing. Both professions are discovering that they have much in common. Oral surgery has probably been one of the greatest factors in bringing about this sympathetic cooperation.—Chalmers J. Lyons, in *J.A.D.A.*, 1935.

NITROUS OXIDE-OXYGEN ANESTHESIA IN OFFICE PRACTICE

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FOR many years after the research findings of Sir Humphrey Davy, nitrous oxide was regarded as a mysterious agent, adopted by the roving mountebank or medical charlatan as a medium of entertainment or to add to the mysticism of their general environment, and its true anesthetic value was not recognized until demonstrated by the efforts of Wells and Morton.

Despite the fact that general anesthesia stems from a dental atmosphere, with brilliant minds devoting a lifetime of energy to the development and refinement of many anesthetic agents, we still have much to hope for. The perfect anesthetic for all patients and for all operations, one that in no way affects the vital processes, which can be used with perfect safety in the infant, the robust youth, the aged and feeble person suffering from arteriosclerosis, the patient affected with a septic condition or the one in shock, has not yet been discovered. In the quest for a satisfactory anesthetic agent, we must first consider the basic requirements, well outlined by Gwathmey.[1]

1. It should be of such nature or combination, or must be capable of such methods of administration, as will reduce the danger of life to a minimum.
2. The anesthetic agent should possess such physical properties, that it is easily taken into the system.
3. It should produce the general, complete or temporary inhibition of action of the nervous mechanism, presiding over cerebration, sensation and motion.
4. It should be capable of administration with the least interference with respiration, circulation, or other vital processes.
5. It should act in such manner that its immediate effects are at all times under control of the administrator.
6. It should reduce the general shock to a minimum.
7. It should not cause serious or lasting after effects, the organisms promptly resuming the physiological functions existing immediately before the administration of the anesthetic.

Today, the anesthetist has command of several agents, which may be used not only alone, but also in mixtures and in sequence, so that for all patients, who necessarily vary in type, age and physical condition, the most suitable anesthetic may be chosen. If, in any field of medicine, there is safety in selection of the means to an end, the domain of anesthesia is that field.

In selecting a general anesthetic that approximates the ideal in the re-

quirements or peculiar demands of dental or oral surgery, especially in its application to office procedures, we must consider other factors besides those enumerated by Gwathmey: availability, rapid induction and recovery, safety in respect to inflammability or explosiveness, convenience of application and odor.

A survey of general anesthetic agents available to date, with a critical analysis of their many qualities, based on the basic factors enumerated by Gwathmey and the peculiar demands of office procedure, leaves much to be desired. Until the ideal anesthetic is discovered, nitrous oxide-oxygen will be the routine office anesthetic of choice when a general anesthetic is indicated.

Nitrous oxide-oxygen offers quick, pleasant induction and rapid recovery with a minimum of postoperative nausea. It is the most suitable general anesthetic for all ages and conditions, with pronounced flexibility, and may be used for short or prolonged anesthesia, with few disadvantages, prominent among which are the cost of the apparatus for administration and the training and experience necessary for proper administration.

In dental or oral surgery, general anesthesia is especially desirable, and it is almost mandatory in many of the acute infections, such as acute septic pericoronitis or cellulitis, pericemental complications and acute osteomyelitis.

In the nervous, uncooperative patient, morbid fear of an operation or of anesthesia is probably the most common handicap. The nervous apprehension incident to any expected pain or physical discomfort is inherent in the majority of patients and, according to Crile,[2] psychic trauma causes reactions comparable to actual pain, making these persons poor subjects for anesthesia. General anesthesia shortens the period of nervous tension.

Children are generally apprehensive and uncooperative when anesthesia is necessary. The unknown always terrifies them and children do not soon forget unpleasant experiences. The child who willingly takes a local anesthetic is the exception to the rule. In extensive extractions, especially in scattered areas, where numerous injections would be necessary, and particularly in inferior molar areas, where bilateral block might be contraindicated, general anesthesia is desirable.

According to Gwathmey[1]: "Nitrous oxide oxygen ranks far above ether or chloroform, so far as safety to life is concerned, and in the point of after effects takes precedence over all other agents, since it is practically free from sequelae, if administered with a fair degree of care." It owes its safety mainly to lack of toxicity and a unique flexibility.

Careful administration of nitrous oxide-oxygen, without symptomatic anoxemia, has no early or late after-effects. The lack of toxicity is probably due, therefore, to the fact that it is an inert gas, anesthesia being produced by purely physical means.

In the blood, acting merely as a highly soluble, indifferent or inert gas,

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it is merely in physical solution and its presence causes no chemical change of any kind—no diminution of the hemoglobin and no effect on the red blood corpuscles.

The symptoms produced in the nervous system by nitrous oxide-oxygen administration are in reality those of a gradually progressing state of anoxemia. The responses of the nervous system occur not as a direct action of the gas, but because of interference with the oxygen supply to its various parts.

The order in which the different parts of the nervous mechanism are affected is determined by their sensitivity and vitality. The brain, the most highly specialized of the nervous tissues, is the first area to react to oxygen deprivation.

Nitrous oxide-oxygen administration is not injurious to and causes no change in lung tissue. It causes no disturbance to the glandular system. The functions of the kidney, liver and other organs are not affected. It has no direct action or effect upon the muscle fibers. The muscle reactions are of nervous origin, frequently inhibitory, induced by the decrease in oxygen intake and varying with the different stages of anesthesia, as reflex movements in light anesthesia, relaxation in surgical anesthesia and rigidity or spasticity in the deeper planes. Its flexibility is ideal in the poorest risks, because the response of the patient to slight changes in the mixture of nitrous oxide and oxygen is so rapid that he can be instantly placed in any desired plane of anesthesia, depressed or lightened, as the situation may require.

Classification of a patient as a subnormal risk should not, in itself, constitute a contraindication for the administration of a general anesthetic, the choice of anesthetic depending largely on the degree of surgical risk and toxicity of the anesthetic agent to be employed. The degree of risk is determined largely by the pathologic process present in the circulatory, respiratory and renal system. Because of its non-toxic nature, nitrous oxide-oxygen becomes the anesthetic of choice for sick patients and a patient too sick to tolerate nitrous oxide-oxygen is too sick for any form of anesthesia. As the degree of risk increases, the necessity becomes greater for observing the reaction of the patient during narcosis and for maintaining the maximum of oxygen in the mixture at all times. With a normal patient, the minimum of oxygen is administered. For a subnormal patient, the maximum is indicated. In the consideration of diagnostic methods for the determination of anesthetic risks, certain evaluation tests may be applied. These tests help in determining the degree of surgical risk and also enable the anesthetist to estimate or anticipate the patient's reactions.

According to McCarthy,[3] tests of vital capacity are probably the most important and efficient means of determining the capacity of the respiratory system, the greatest hazard of nitrous oxide-oxygen anesthesia being inefficient respiration. This is much more important than cardiac pathosis,

for even if the heart is damaged, satisfactory anesthesia can be maintained if ventilation remains adequate. However, if respiratory obstruction occurs or if some intrathoracic pathosis limits respiratory exchange, even in a patient with normal circulation function may be seriously embarrassed, and with an impaired myocardium, the heart action may be badly depressed. Determination of vital capacity before anesthesia will, in many cases, be the only means of detecting an impaired or embarrassed respiration, and it is a test that could be used more commonly to great advantage.

One single test recommended by authorities for the great majority of patients is Henderson's[4] breath-holding test, the results of which are dependent on the size and age of the patient and his physical activities and the condition of the lungs. To make the test, the patient lies absolutely quiet for five minutes. He then takes a moderately long breath and holds it as long as possible, lips closed and nostrils pinched together. A normal subject should be able to hold his breath under these conditions for forty-five seconds. Thirty seconds would indicate good vital capacity, progressively decreasing to twelve or ten. Below this, gravely impaired vital capacity is indicated, with possible cardiac involvement, and such a patient would be classed as subnormal.

Free breathing requires the constant attention of the anesthetist, since any interference with respiration restricts absorption of gas by the blood, increases the accumulation of carbon dioxide and causes difficult breathing. This leads to anoxemia, which implies an intake of oxygen insufficient to maintain the vital functions of the heart, lungs, etc., an intake below the oxygen threshold for the patient, with cyanosis, occurring at the same time. The presence of cyanosis means that there is insufficient oxygen intake to convert all the hemoglobin in the circulating blood to oxyhemoglobin. The significance of cyanosis varies with the type of patient, robust, plethoric or anemic. Cyanosis occurs usually during the induction stage, and symptomatic anoxemia after the anesthesia has progressed somewhat. The presence of cyanosis may be no evidence of danger, and its absence no sign of safety.

To quote McKesson[5]: The administration of gases under slight pressure is an important aid and safeguard to the patient's breathing, as the inspiratory phase is much weaker than the expiratory, and, further, because obstructions in the airway tend to become more marked with inspiration than with expiration. Obstruction increases in varying degrees, the load on the muscular elements of the respiratory system thus tending to exhaustion. Even the partial obstruction unduly prolonged may lead to a fatal termination from asphyxia, anoxemia and exhaustion.

Some causes of obstruction are malposition of the patient's head, flexion of the head on the chest or blocking of the throat by the tongue, when the chin is dropped. Pressure on the tongue or lower jaw and the presence of

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blood and mucus are the most common causes, with occasional difficulty from misplacement or loss of control of the mouth packs.

Less frequent causes of obstruction are growths in the throat, trachea or bronchus. The treatment is largely prevention.

In deviations from the normal, the use of artificial airways is a great aid in correcting respiratory embarrassment.

In dental anesthesia, the use of nasopharyngeal tubes made of rubber assists greatly in maintenance of an open airway. These are used with ordinary nasal inhalers. They vary in length and diameter to fit various sizes of nares and should be long enough to reach well down into the nasopharynx, beyond the base of the tongue and epiglottis. The tube is curved and one end is beveled to facilitate insertion.

A tube, covered with some form of lubricant, is introduced along the floor of the nose, the long side of the beveled end toward the septum, with care to avoid nasal bleeding. This procedure should follow the induction period, when the patient is unconscious. The tube is removed before the patient returns to consciousness.

One important adjunct to efficient anesthesia is adequate premedication. Within the last few years, we have found it practically indispensable, routinely using one of the drugs in the barbiturate series. Premedication has a twofold effect, first physical and second psychic.

According to Clement, [6] "The physical effect of preanesthetic medication decreases the general body metabolism or lowers the demand of tissues for oxygen, and raises the threshold of irritability for both carbon dioxide and oxygen want."

The reduced metabolism results in a slower heart rate, with less fluctuation of the blood pressure and a decrease of muscular activity generally throughout the body. A slower pulse means less cardiac labor, less exhaustion and therefore less depression.

The psychic effect of premedication is to lessen or abolish the patient's fear and apprehension and safeguard him from psychic shock. A beneficial effect on the endocrine activity is a lessening of the secretion of thyrotoxin from the thyroid and of epinephrine from the adrenals. Both ordinarily are increased by the state of fear and excitement.

Skill and experience plus unrelaxing vigilance are essential.

A patient submitting to anesthesia literally places his life in the hands of the anesthetist, and the administration should never be considered lightly. Successful anesthesia must vary in direct proportion to the skill and experience of the operator.

To quote Abernathy:

> In the administration of a general anesthetic, the exercise of judgment and skill is necessary and this can be obtained only through special study and experience. As much care should be used in the administration as in the per-

formance of the operation, and the anesthetist should have, and does have, in properly regulated cases, as much responsibility as the surgeon.

Emphasized in the foregoing article is the importance of the rôle of the anesthetist as a distinct personality in contradistinction to the person who essays the double rôle of operator and anesthetist or director of anesthesia, his eyes and hands alternating between patient and gas machine. Then comes the complication of a poorly balanced anesthesia, and a struggling patient with nausea, plus operative difficulties demanding the employment of cheek distenders, tongue depressers, chisels, burs or a suction apparatus.

Picture the dilemma: The mental reaction which destroys the balance and finesse so necessary to successful surgical procedure and the liability to bodily injury or loss of the patient from overdosage.

I subscribe to the desirability, if not the necessity, of having available the services of a well-trained anesthetist, supplemented by those of a surgical assistant, the latter to relieve the anesthetist from any concern with the progress or outcome of the operation, and to make possible the successful conduct of delicate or extensive operations. The safety of nitrous oxide-oxygen anesthesia is, of course, only relative. No anesthetic agent is any safer than the person who employs it, and there is no doubt that, because of the rapidity with which the patient passes from one plane to another, the administration of nitrous oxide-oxygen presents difficulties with which the inexperienced or untrained person is not equipped to cope.

Since all dependable statistics and clinical observations establish the high status of nitrous oxide-oxygen anesthesia, possessing, as it does, all the features desirable in office procedures in oral surgery, it is urgent that those wishing to avail themselves of the many qualities of this valuable anesthetic shall perfect their technic and so further the effort toward alleviation of pain.

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THE DRY SOCKET PROBLEM

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THERE is no phase of dentistry that presents a greater problem than the control and alleviation of postoperative extraction pain. Meticulous as the operator may be, the occurrence of this malady is occasional in all surgical practices. Nothing mars an otherwise smooth operative day so much as a patient or two with so-called dry socket. Too many operators dismiss the patient without thorough examination and proper local treatment. Too many fail to diagnose this painful condition in its true form, and frequently send the patient away with a free package of some proprietary, shot-gun remedy, telling him, as Archer[1] reminds us, that he "must have caught cold in his jaw." It appears that the actual condition is frequently unrecognized. When a patient returns with a complaint of severe reflex and neuralgic pain four or five days after an extraction, detailed examination of the socket should always be made. The dentist should take a small curet and pass it carefully through the rather tough blood clot to determine whether a dry socket is present. Invariably, the bone is found to be denuded and the "feel" of normal healing is noticeably absent. This, together with the patient's description of his symptoms, should indicate the existence of postoperative pathosis. The term dry socket, like "trench mouth" and "mumps," permits the laity to understand what the condition actually is and needs no elaborate explanation. In these crowded days, then, why make any attempt to replace the term dry socket with a more pretentious terminology? Archer[1] has recommended that the condition be described as alveolalgia. Whether or not this new word is accepted in dental nomenclature, the condition will still be "dry socket" to most patients and dentists alike.

A perusal of the literature on the subject of dry socket reveals little to indicate a common understanding and rational treatment of the condition.

Armstrong[2] makes the significant statement that " 'dry socket' actually exists before the tooth is extracted, but manifests itself only after the irritation of extraction has occurred." I know of no better way to clearly and concisely state the entire fact. I believe that more than half of the dry socket cases can be anticipated. In these instances, sedative dressings should be inserted immediately after the extraction so that discomfort and painful neuralgic reflexes can be prevented. Such a procedure naturally would be based on a case study of the roentgenogram, the clinical appearance of the mouth and the general physical condition, together with an understanding of these factors. It is much easier to prevent than to cure. (Fig. 1.)

The important factor in my opinion is general resistance. A goodly number of the patients coming in for treatment are under a physician's

care for systemic ailments, and their response to extraction and other surgical procedures cannot always be without complication. Let us be through with taking the defensive, cooking up excuses and assuming full blame for the postoperative occurrence of pain. We can do no better work than the material we have to work on permits, and I make this statement emphatically to the complaining patient in these dry socket cases.

Durbeck[3] says:

> The occasional case of extreme postoperative pain of prolonged duration may rightfully be considered one of the most distressing postoperative sequelae. Its usual combination with necrosis and putrefaction of the blood clot constitutes the so-called "dry socket." This misnomer, while generally used and accepted, is actually a *highly localized osteitis* over which the operator has little or no control as to prevention. Essentially, the condition is an idiopathic one and, inasmuch as its etiology is quite unknown, its occurrence can never be considered as a reflection on the ability, skill, or judgment of the surgeon. . . .

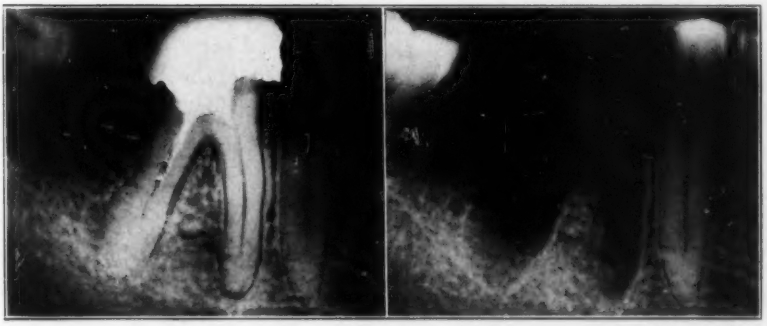

Fig. 1.—Case of dry socket; showing lack of contrast between distal root and bone.

The diagnosis of the dry socket is entirely clinical. The patient invariably complains of an extremely fetid taste, much more foul than the usual unpleasant taste of the early postoperative period. Pain is usually severe and frequently is felt in the region of the ear as well as in the socket. It is of a radiating type and is often described as a throbbing or pounding sensation. Inspection discloses that the blood clot is either entirely spent or is badly broken down, although a casual observation often fails to elicit this information. A blunt, flexible probe when introduced will meet with little or no resistance and will cause increased pain if allowed to touch the socket wall. Frequently the remaining portion of clot presents a dirty greyish cast rather than the dark red appearance of the normal one. Macroscopical pus usually cannot be demonstrated.

Molt[4] brings out the importance of diet as having a direct bearing on the patient's general resistance. It is his observation that dry socket, so-called, has been a vexing problem to the dental profession for years. Many theories

have been offered as to its causation, chiefly (1) the use of a local anesthetic, (2) the washing out of the blood clot and (3) the introduction of infection into the wound. None of these seems completely tenable, as (1) such a condition is as frequently found after extraction under a general anesthetic; (2) dry socket occurs when no rinsing has been done and where a firm and satisfactory blood clot has been formed, but later disintegrated, and (3) the condition frequently occurs although the utmost precaution has been observed as to asepsis in operating. True, smears from the walls of such a socket show a multiplicity of organisms, with the fusiform bacillus and spirochete of Vincent in great numbers, but these organisms are known to be great scavengers and may be found in equal number in a tonsillectomized area or any exposed incised surface in any mouth a day or two after operation.

As the mechanics of bone deposition in tooth sockets are identical with those of fracture healing or redeposition in osteomyelitis, it seems quite prob-

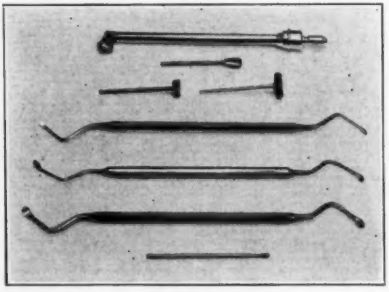

Fig. 2.—Top to bottom: Intra-oral right angle light for detailed examination and surgical work; trimming bur for "saucering" third molar crypts; circular bone files to smooth jagged alveolar process; sharp knife-edged curets; bone bur.

able that, at least in a large proportion of cases, the same basic contributory factor is present.

We have been going through a period in which the most absurd dietary restrictions, offered by just about anyone, no matter what his qualifications, have been accepted and utilized. Reducing diets have been responsible for the complete loss of health in a multitude of cases. Diets have been prescribed for patients suffering from various diseases, such as gastric ulcer, colitis and cholecystitis, with no regard for any part of the body other than that directly concerned.

The majority of patients for extraction are taken, we might say, literally "off the street." They come in because they have toothache or because, for some reason or other, and possibly without the advice of a physician, they have decided that certain teeth should be removed. A satisfactory and complete evaluation of the patient's physical condition is, under such circumstances,

hardly possible. An undernourished patient is a poor risk for any operation, and that applies quite as definitely to minor as to major surgery. Undernourishment may be present, of course, for reasons other than deficiencies of diet, but the most impressive of all the cases under my observation have been the dietary deficiency cases.[4]

It has been my experience that a large percentage of postoperative difficulties occur in undernourished patients. The young girl of today, subsisting largely on a "cigaret and cola" diet, comes to mind in this regard.

The contributing causes of postoperative pain are chiefly the anesthetic, traumatism and sepsis.

1. In using local anesthetics, attention must be given to the method of administration, the condition of the syringe and needle and the type and concentration of the solution used. It should be injected very slowly so that the tissue takes it up without death of some of the cells ensuing. It should be strictly aseptic, isotonic, freshly sterilized and warm. There is usually less postoperative reaction when the anesthetic is injected at some distance

Fig. 3.—Equipment for routine treatment of average case of dry socket: gauze; drugs; irrigation cup; half-ounce jar for making up dressings; cotton pliers; eustachian catheter and bulb; insertion fork.

from the site of operation. This definitely favors the conduction method, which should always be employed in the mandible, where the more severe types of pain usually occur.

2. Trauma might be termed a possible contributing factor in dry socket. Direct injury to the tissues involved always results in a variable amount of cell deterioration. Aside from general health differences, then, it is accurate to assume that the degree of postoperative reaction is directly proportionate to the amount of injury. Excessive pain may be caused by exposure of the alveolar process, the presence of jagged edges and splinters of bone or dislodgment of filling material. Great care must be taken to see that the wound is in the best possible condition before the patient is dismissed after an extraction. The rough bone must be smoothed, loose particles of alveolar

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process and foreign substances must be removed from the tooth socket, and an effort must be made to have the mucous membrane cover raw bone surfaces as nearly as possible. Therefore, the use of sharply cut flaps and sutures is to be encouraged in all complicated extractions.

3. A condition of sepsis alone may cause trouble. Organisms may become dominant in the area of lowered resistance and marked inflammatory reactions result. It is a revelation to find what strict asepsis will do to reduce after-pain and the incidence of dry socket, because, as a rule, the organisms introduced upon instruments or in the anesthetic are responsible for the reaction, rather than the organisms existing in the mouth before operation.

Dry sockets may be classified as follows: (1) the socket that is completely denuded of blood clot, (2) the socket filled with necrotic granulation and (3) the socket the orifice of which is covered over by a rather tough, degenerated epithelial cap.

Fig. 4.—Paste type dressing; showing syringe and tip used in its introduction.

TREATMENT

To treat dry socket successfully, care must be exercised to get to the source of the trouble. A brief medical history should be taken, and if the patient has had a physical examination recently, the examining physician should be consulted. Systemic infection has been previously mentioned as an important factor in the condition. Tuberculosis, rheumatic fever, syphilis, diabetes, chronic colitis, asthma, pregnancy, glandular deficiencies—these are but a few of the conditions to be considered. The emotional stability of the patient also plays a part. The hysterical and neurasthenic types share in the picture. With proper determination of the background, an approach to the problem has been made.

A thorough study of local conditions must follow. The lazy method of administering sedatives internally and hypodermically without first observing the local conditions is to be condemned. The socket must be examined carefully for possible disturbances therein. The jagged edges of alveolar

process must be trimmed away and all spicules and larger fragments of bone that have been partially or completely detached from the periosteum must be removed. (Fig. 2.) Curettement is contraindicated unless the pain is due to some definite intra-alveolar irritant. It is a most pernicious and destructive procedure when practiced blindly, and is sure to bring on more pronounced complications. Some men ignorantly curet a dry painful socket with the hope of reestablishing a healthy blood clot. If a normal blood clot does not hold primarily, the condition of the surrounding bone is responsible. This bone, having been associated with an infected tooth for years, cannot always be expected to heal normally, and attempting to "rebleed" the socket avails nothing, more severe pain resulting.

I tell patients that dry socket is to be expected in many instances. What surprises me most is that it does not occur more frequently. When, let us say, an infected lower molar, pulpless and diseased for years, has finally been removed, can one assume that the surrounding bone is capable of normal and uncomplicated healing? The socket needs local aid to facilitate cicatrization, and a new blood clot is not at all essential. This can easily be demonstrated in the healing process in radicular bone cysts. Once and for all, then, a blood clot is not necessary and one should not mercilessly curet to make conditions worse.

The local treatment of dry socket consists simply of applying sedative drugs to the denuded bone areas to desensitize the excruciatingly painful nerve endings and to aid in the deposit of normal granulation tissue. After the diagnosis of dry socket has been established, the wound should be carefully irrigated with a warm saline solution, preferably by means of a eustachian catheter with a rubber ear syringe. With the catheter tip, the amount and extent of denuded bone and the area to be covered can be determined. A roentgenogram should be made of the socket to make doubly certain that there are no irritating foreign substances therein. In the event that a chip of tooth structure, spicule of detached bone or bit of foreign material is recognized, a small blade-edged curet is deftly introduced and the offending irritant removed. A dressing is now prepared and carefully introduced to completely protect the raw bone wall. Various drugs and various types of paste are used for this purpose. For years, I have depended on a one-quarter or one half inch wide gauze strip into which is incorporated a liquid made up of 40 per cent chlorbutanol in oil of cloves, oil of wintergreen and oil of cassia, together with benzocaine powder.

A dressing should never be packed, but should be loosely placed. The tip of the dressing, however, should be carried to the apical end of the socket, and all raw, sensitive bone should be gently covered. This treatment will take care of the average case. The dressing should be removed in forty-eight hours, the socket again irrigated with physiologic solution of sodium chloride, and dressings inserted for progressively shorter periods every two or three days until healthy granulation begins. The

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patient should report complete comfort for at least four days before discontinuance of the gauze strip dressings. After the patient has reported complete comfort for a period of several days, and granulation is found to be satisfactory, I insert a mild sedative paste, which covers sensitive tissues until they have become accustomed to exposure to air and mouth fluids. (Figs. 3 and 4.)

In the more complicated cases, heroic treatment should be instituted. If a patient returns in from five to eight or ten days after extraction with the report that he has experienced great discomfort for several days, the socket will have to be handled more delicately. If the wound is found so sensitive that even irrigation is painful, a short strip of one-half inch gauze thoroughly moistened with 2 per cent pontocaine hydrochloride should be boldly and immediately introduced into the socket. This should be allowed to remain for from twenty to thirty minutes, after which anesthesia will be sufficient for insertion of a sedative dressing. In extreme cases, local anesthesia may be advisable. The pontocaine hydrochloride pack having been removed, the wound is irrigated and the sedative dressing inserted. At times, the addition of 10 per cent butyn (sulfate topical anesthetic) will increase the potency of the dressing. Then, too, in certain large lower jaw wounds, where contamination and dilution by the saliva seem to interfere with successful treatment, the regular sedative dressing is introduced into the apical two-thirds of the socket, and a paste compound of zinc oxide and engenol is placed over the dressing to seal the socket from the mouth fluids. Zinc oxide-eugenol alone is many times very effective, and if satisfactory results are obtained with the first treatment, it can be allowed to remain for several days.

Comenting on this type of treatment, Levine[5] says:

> The annoyance of "dry socket" is now a thing of the past since I adopted the use of a sedative pack; in most cases only one treatment is required. To date, I have never had to insert a third pack, and only in a few cases a second one, the pain stops instantly and never returns and the medicated pack is gradually pushed out as the socket fills with granulation tissue. Not only is the pain instantly relieved, but the vile odor and taste of which patients complain disappear almost at once.
>
> For treatment I use a paste composed of zinc oxide and eugenol mixed somewhat softer than that used for a cavity base; softness and plasticity are of prime importance. This paste is used as a pack which constitutes the entire treatment.
>
> The socket is washed out with any warm, bland solution and then wiped gently with a pledget of cotton. A cone of the paste is molded to the approximate size of the socket to form the pack. The apical third is then cut off and the shortened pack is coaxed gently into the socket.
>
> It is a good plan to have the patient return in about four days and remove the pack. Following removal of the pack, irrigate the socket gently with a warm, bland solution and dismiss the patient.

Should the patient not return, however, no harm will be done, since the pack will eventually come out of its own accord as the socket heals.

Paul E. Allen reports excellent results with a paste made up of iodoform, 8 gm., eugenol (or oil of cloves), 4 cc. and carbolated vaselin to make 45 gm. He says:

> In lower third molar impactions, after delivery of the tooth and the toilet of the cavity, insert sutures but do not tie them. Fill the socket with the above-detailed paste dressing and close the flap. The sutures then can be tied and cut. This dressing melts to liquid at mouth temperature and will find its way out as a normal blood clot forms below. Being in a vaselin base, it will not in any way interfere with the blood clot and normal healing; yet, while it is there, its combination of antiseptic, analgesic and counterirritant action will alleviate all pain in most cases. Do not remove.

In a rare instance, a socket refuses to heal. The dressings are not particularly effective, and there is a retarded and sluggish type of tissue observed. In these cases, I recommend cauterization of the entire socket, using a 50 per cent solution of trichloroacetic acid. A short length of one-half-inch gauze is moistened with the acid and quickly and deftly the socket is wiped out. Irrigation should follow, after which the usual sedative dressing is introduced.

Impacted lower third molar wounds should, I believe, be treated similarly to routine dry sockets. I have heard a few operators say that they can remove deeply impacted teeth and suture completely and, by spraying the wounds with sulfonamides, effect primary healing without pain. This has not been my experience.

A discussion of the dry socket problem would be incomplete, however, without consideration of the sulfonamides. After all rather unusual extraction cases, and particularly in the case of complicated surgical removal, I immediately spray the socket lightly with a mixture of one-third sulfanilamide and two-thirds sulfathiazole powders. To facilitate its introduction, I use a powder blower attached to a regular compressed air outlet. Then, without permitting the patient to expectorate, I at once place a small folded exodontia sponge over the wound and instruct the patient to close the teeth over this. On dismissal, the patient is instructed to keep the gauze sponge in place for about half an hour, and then not to rinse the mouth for five or six hours.

Millhon *et al.*, in an attempt to evaluate the sulfonamides in the prevention or treatment of dry socket, have failed to present conclusive evidence that importance can be attached to the employment of these drugs. In summarizing the article, they say: "Sulfathiazole alone does not prevent 'dry socket.' We have not found sulfathiazole of value in the treatment of 'dry socket' once symptoms have developed." In a tabular comparison of the incidence of "dry socket" after the removal of impacted lower third molar teeth, their results show that, in 149 cases in which medicated and

unmedicated gauze dressings were used, there was an incidence of 3.4 per cent, and, in a series of 397 cases in which 60 per cent sulfathiazole in glycerin base was used, 2.8 per cent dry sockets developed. These figures to my mind do not demonstrate any advantage in the use of sulfathiazole over the use of medicated gauze dressings in follow-up treatment.

Postoperative instructions to the patient are paramount. He should be fully informed of the importance of keeping the mouth clean. A toothbrush should be used and the teeth and tongue cleaned regularly, to prevent the deposit of mucous débris. After extraction, it is difficult for the patient to comprehend and remember instructions given by word of mouth. For this reason, I use typed forms to stress the most important points to be observed in each case. Only in this way can a complete, satisfactory service be rendered.

SUMMARY

1. Dry sockets are a problem in most dental practices.

2. The conditions which cause dry socket are present before extraction and, in most cases, can be recognized.

3. The physical condition of the patient is a primary factor in dry socket.

4. Contributing etiologic factors are anesthesia, trauma and sepsis.

5. Treatment should be systemic and local.

6. Curettage is usually contraindicated in treatment of dry socket except when pain is seen to be due to some definite intra-alveolar irritant, which must be removed or enucleated.

7. In the local treatment, sedative drugs of proved value are essential. The sulfonamides are being used experimentally in some cases, but their merit has not yet been definitely established.

8. Impacted lower third molar wounds should be treated similarly to routine dry socket cases.

9. Postoperative instructions to every patient are of paramount importance.

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HISTOPLASMOSIS: REPORT OF CASE

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WITH the continued rise in incidence of histoplasmosis, the oral diagnostician should consider this condition in the differential diagnosis of cases of intractable ulceration of the oral cavity, and should be sufficiently well versed in its clinicopathologic picture to recognize its characteristic features and to utilize definite diagnostic studies. Although the disease has been looked upon as relatively a rarity, the increase in number of reported cases may possibly be attributed to a more general knowledge of the disease rather than to an increase in the frequency of occurrence.

Histoplasmosis was first recognized in 1905, when Darling, searching for visceral and cutaneous leishmaniasis in the Panama Canal Zone, found three cases of cutaneous infection which appeared similar to kala-azar. He believed that the organism found in the tissues of these patients was a protozoan and called it *Histoplasma capsulatum*. It was not until 1912 that Rocha-Lima, studying the bacteriology of the disease, concluded that the organism was a cryptococcus, one of the fungi. Medical literature reveals only seven reported cases in the succeeding twenty-two years, but the case reports since 1934 have somewhat increased in number.

Despite its so-called low incidence, it is evident that the disease is widespread. Geographic distribution of case reports has included Panama, the Philippines, Argentina, Brazil, Honduras, Java and at least fourteen states of the United States, Michigan perhaps reporting as many as or more than any other state. The epidemiology and pathogenesis of the disease warrant further investigation. The frequency of oral and upper respiratory tract involvement noted in the cases reported suggests that these areas are a frequent site of initial infection.

The common clinical characteristics are splenohepatomegaly, lymphadenopathy, pyrexia, leukopenia, anemia and, in some cases, chronic respiratory disease. Accumulated data reveal that the clinical features of the disease are subject to considerable variation. Liver and spleen enlargement, anemia or leukopenia may be absent. A few authors report a leukocytosis, which is perhaps due to secondary infection.

Pathologically, the most important symptom is reticulo-endothelial proliferation, especially in the spleen, lymph nodes, liver and bone marrow. Blood smears may reveal the parasites phagoctosed in the large mononuclear cells and occasionally in the neutrophilic polymorphonuclear leukocytes.

From the Department of Oral Surgery, University Hospital, University of Michigan.

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Definite diagnosis is dependent entirely on laboratory data. The small (1-3 micron) oval encapsulated organism must be demonstrated. It may be found in the mononuclear cells in peripheral blood smears, sternal bone marrow smears, lymph nodes or spleen removed at splenectomy and in biopsy sections of cutaneous or mucosal lesions.

Therapy up to the present time has been unsatisfactory, and mortality 100 per cent. The drugs most frequently suggested for treatment are antimony compounds. Therapeutic doses of ionized copper, potassium iodide and local radiation therapy have been tried without avail. One case has been cited in which clinical recovery was noted after treatment with neostam, an antimony compound. To date, the majority of diagnoses have been postmortem. Increased knowledge of the disease will lead to its being more generally recognized, with the ultimate possibility of a specific therapy.

Shallow ulcer on dorsum and margin of tongue.

REPORT OF CASE

History.—A man, aged 59, entered the University Hospital January 18, 1943, complaining of sores on the tongue and upper lip. The lesions had had their onset four months before admission in the form of a small painful ulcer on the tongue. Within a few days, the mucous membrane of the upper lip was involved by a similar lesion. Increase in size was minimal. Exquisite tenderness was the prevailing symptom, and intake of food had been interfered with as a result. Weakness and fatigue followed.

The patient had been a farmer nearly all of his life, a native of Michigan, and had never resided outside the state. For many years, he had been a heavy tobacco chewer, and he had the habit of chewing bits of grain and straw that

were picked up from the fields. There was nothing in the medical history that appeared definitely contributory to the present illness.

Examination.—The patient was well-developed, though emaciated, and appeared acutely ill. The temperature was 101° F.; pulse, 60; respiration, 18; blood pressure, 100/60. There was moderate diffuse swelling of the upper lip. Intra-oral examination revealed an irregularly circumscribed shallow ulcerative granulomatous lesion with undermined borders on the left dorsum and lateral border of the tongue, as shown in the illustration. The involved area measured 2.0 by 2.5 cm. A similar ulcer on the mucosal surface of the upper lip measured 4.0 by 1.5 cm. A barely palpable left submaxillary adenopathy was noted, but complete physical examination failed to reveal other significant conditions. The kidneys, liver and spleen were not palpable.

Laboratory Data.—On admission, the red blood cells numbered 3,900,000; white blood cells, 6,200. Hemoglobin was 63 per cent (9.8 gm.); polymorphonuclear neutrophils, 74.5 per cent; large lymphocytes, 12.0 per cent; small lymphocytes, 2.0 per cent; monocytes, 10.0 per cent; eosinophils, 0.5 per cent; basophils, 1.0 per cent. The platelets were slightly increased in number. The hematocrit reading was 32.5; mean corpuscular volume, 84; sedimentation rate, 28 mm. per hour. A diagnosis of hypochromic normocytic anemia was in order. Repeated blood studies over a period of five months did not demonstrate significant fluctuation.

Urine examination, including special studies for tubercle bacilli, was negative. Repeated serologic tests, both Kahn and Kline, including spinal fluid examinations, were negative. Multiple sputum and stool examinations and cultures revealed nothing significant. Late in the course of the disease, a fungus-like organism was seen in the sputum, but the organism could not be identified by the usual bacteriologic methods. Biopsy material from the oral lesions was cultured, and the material was inoculated into laboratory animals, but the cultures did not reveal unusual organisms, nor did the inoculations produce disease. Direct smears were likewise not significant.

Further bacteriologic studies included blood cultures, many taken at the height of febrile reactions and immediately after the onset of chills. Blood so obtained was injected into laboratory animals. Direct smears were examined for malaria. These results were persistently negative. Careful examination, particularly of the monocytes, was made of sternal puncture specimens and centrifuged venous blood, but organisms were not detected. Phagocytic activity was not remarkable, and agglutination was negative for undulant fever and tularemia. With antigens in varying dilutions, intradermal skin tests were made for tuberculosis, coccidiosis, blastomycosis and histoplasmosis, but the reactions were negative. The value of the histoplasma antigen was questioned at the time of its use.

Roentgenographic Findings.—Retrograde pyelograms made ten days after admission were negative. Chest films taken at the same time revealed a lesion in the right upper lung field (identity indeterminate), emphysema and a senescent aorta. Examination one month later demonstrated no perceptible change, but two months later, a pneumonitis was noted in the right base. Ten days after the last film was taken, the pneumonitis appeared to be subsiding, but a bilateral generalized fine granularity was noted throughout both lungs.

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Chest films, taken April 22, three months after admission, revealed complete resolution of the process in the right base, but a new focus of parenchymal infiltration was seen in the left base. The possibility of a virus pneumonia was considered.

Course.—Throughout hospitalization, the oral lesions persisted without appreciable change. Pharyngeal and laryngeal lesions of a similar character developed, with hoarseness and cough. There was remittent septic fever, at times exceeding 104° F. orally, with increasing manifestations of systemic disease in the form of excessive fatigue, weakness and loss of weight. The febrile reactions were usually ushered in by violent chills. Recurring attacks of dyspnea were thought to be manifestations of laryngeal edema.

All lesions were repeatedly submitted to biopsy. Each microscopic examination of the material obtained revealed a chronic infectious granuloma without caseation and with occasional multinucleate giant cells. The specimens presented characteristics suggestive of both syphilis and tuberculosis. In sections stained by the Giemsa method, numerous small yeastlike inclusions, chiefly in the reticulo-endothelial cells, were noted. They could not be definitely identified as any known pathogenic organism and their significance could not be determined. The close resemblance of the disease process to the classic picture of histoplasmosis was constantly considered despite failure to substantiate our opinion by known histopathologic and bacteriologic methods.

Treatment.—The retrogressive course demanded desperate empiric measures. At varying intervals, the patient received iodides, bismuth, antimony and neostam, in addition to sulfanilamide, sulfathiazole and sulfadiazine in full therapeutic dosage for trial periods. Germicidal and parasiticidal solutions were applied topically. The diet was heavily fortified with iron and vitamin concentrates. Radiation therapy was instituted as a trial therapeutic agent, and the patient received 900 roentgen units to the lip, face and neck fields. He was temporarily discharged on the seventy-second day in hospital after exhaustive diagnostic studies and therapeutic measures.

Readmission.—The patient was readmitted one month later. There was increased hoarseness and a severe hacking cough. There had been progressive dysphagia with a weight loss of 30 pounds since the discharge from hospital. Upper gastro-intestinal roentgenograms revealed a high esophagotracheal fistula, and a gastrostomy was performed. Chills and fever continued. Bismuth, antimony and iodide therapy was again instituted. Multiple whole blood transfusions were administered.

Approximately six weeks after the second admission, the patient suddenly developed cyanosis and an electrocardiogram was made. The heart rate was 193; the P-R interval, 0.15; the QRS, 0.08. Attacks of paroxysmal tachycardia with prominent variation of form of the electrocardiogram and conspicuous irregularity of rhythm were observed. There were single extrasystoles between attacks and very marked prolongation of the Q-T interval.

July 16, six months after the first admission and ten months after the onset of symptoms, death ensued from cardiac failure.

Necropsy.—A postmortem examination revealed the cause of death as subacute verrucous and ulcerative tricuspid endocarditis.

Gross findings: The complete gross pathologic diagnosis was as follows:

chronic (mycotic?) ulcer of the tongue; subacute (mycotic?) granulomatous lesions of the hypopharynx, base of tongue and larynx with ulceration and destruction of true and false vocal cords; subacute (mycotic?) verrucous and ulcerative tricuspid endocarditis; subepicardial fatty infiltration, myocardial fatty infiltration and myocardial degenerative infiltration; early arteriosclerotic nephropathy with degenerative fatty infiltration of the renal pyramids; moderate bilateral apical and right interlobar pulmonary adhesions; fibrotic Ghon tubercle; localized atelectasis of right base; esophageal leukoplakia; early focal subcapsular hemorrhage in left lobe of liver; moderate acute passive congestion of spleen; dropsy of choroid plexus; external hemorrhoids.

Microscopic findings: *Histoplasma capsulatum* in small number was found in the adventitia of the smaller blood vessels in the cortex of the brain and in a few mononuclear phagocytes in the leptomeninges. Giemsa stained sections showed colonies of the organisms in a small ulcer on the auricular surface of the tricuspid valve and in the subendothelial connective tissue. The aorta, Giemsa stained, showed the organisms in the media and all appeared intracellular. In the lungs, the organisms were identified in an occasional mononuclear phagocyte and giant cell in the fibrous connective tissue in the interalveolar septa. No organisms were seen in the bronchial lymph nodes in the Giemsa and Masson trichrome stained sections. *Histoplasma capsulatum* was demonstrated in an occasional large mononuclear phagocyte in the granulation tissue at the base of the ulcer of the tongue by Giemsa and Masson trichrome technic. An ulcer at the base of the epiglottis in the Giemsa preparation showed an occasional *Histoplasma capsulatum* in plasma cells, lymphocytes and giant cells. Many of the organisms were seen in the lymphocytes, plasma cells and large mononuclear phagocytes in the granulomatous tissue of the mucosa of the larynx with Giemsa stain.

Large mononuclear phagocytes in the red pulp of the spleen contained what appeared to be shadows of *Histoplasma capsulatum*. However, no organism was definitely identified as *Histoplasma capsulatum* in the Giemsa and Masson stained sections.

The liver showed four bodies resembling *Histoplasma capsulatum,* but Giemsa and Masson stain showed no organisms.

In the ureters, Giemsa-stained sections showed two *Histoplasma capsulatum* organisms in a macrophage among the lymphocytes in the walls.

SUMMARY AND COMMENT

The incidence of histoplasmosis, a fatal disease caused by the specific fungus *Histoplasma capsulatum,* has risen sharply. It can no longer be considered a rarity, but must be ruled out in any condition characterized by painful progressive non-healing, chronic ulcerative granulomatous lesions associated with enlarged spleen and liver, leukopenia, anemia, lymphadenopathy, pyrexia and chronic respiratory disease. Observation of persistent ulcerative granulomatous lesions of mucous membrane surfaces should always suggest the possibility of histoplasmosis.

Since oral ulcerations are a common finding and the oral diagnostician is the person most likely to be confronted with this disease as a diagnostic

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problem, it should be added to his list of lesions in differential diagnosis. The case of histoplasmosis here presented had oral lesions as its sole significant manifestation until the terminal stages.

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Cleft Palate.—Intelligent instruction in the art of speaking correctly should always follow the operation. Training will do more for improvement of speech than will the operation. In these cases, speech is always defective in three important particulars; namely, resonance, melody and articulation. Resonance and melody of the voice cannot successfully be restored by the operation alone on account of the limited control that results from the palate's being more tense than normal, owing to the operation. The tenseness of the palate will prevent the regulation of the size of the opening between the oral and nasal pharynx, which is constantly changing in the process of phonation. The nasal resonance will depend largely on the ability of the patient to close this opening. The melody of the voice depends largely on the functions of the pharyngeal muscles, and unless these muscles are placed in their normal position, normal melody of the voice will be difficult to obtain.—Chalmers J. Lyons, in *J.A.D.A.*, 1930.

ACTINOMYCOSIS: REPORT OF CASES

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CASE 1.—*History.*—H. H., a man, aged 20, a student, had had both lower third molars removed under local anesthesia five weeks before. The immediate postoperative recovery was uneventful.

Four weeks later, there was an onset of soreness and swelling on the right side of the face, which became progressively worse. The patient returned to his dentist, who referred him for consultation and treatment.

Examination.—April 3, 1939, examination revealed a tumescence along the right horizontal ramus of the mandible. No area of fluctuation could be discerned. The intra-oral examination revealed that both lower third molar sockets had healed. The general condition of the mucous membranes and teeth was excellent. Dental and lateral jaw roentgenographic examinations were negative for osseous or dental disease.

Treatment and Progress.—Supportive measures, with continuous application of hot wet compresses and mild sedation for control of pain, were prescribed. The expected pointing was slow. However, April 14, an area of fluctuation was discernible along the midportion of the right horizontal ramus of the mandible. An extra-oral incision and drainage were carried out under gas anesthesia. Satisfactory drainage of a large amount of foul purulent discharge was obtained. A rubber drain was inserted. There was relief of symptoms for only a few days, then recurrence of pain and swelling.

By April 25, an area of fluctuation was present just below the zygoma. There was little drainage from the original incision. The skin over the involved areas were a dusky purplish red. Suspecting actinomycosis at this time, I reviewed the history with this in mind. Two or three days after the teeth were removed, the patient went on a hayride and he recalled having chewed on a straw during part of the ride.

April 25, drainage was accomplished by inserting a large bore needle into the fluctuant area below the zygoma and aspirating the pus into a syringe. This was done under gas anesthesia. Sulfur granules were found which showed the ray fungus of actinomycosis on microscopic examination.

In consultation with Dr. Ferris Smith, it was decided to give the patient an increasing dose of thymol crystals and x-ray therapy. Fifteen grains was given daily; 5 grains in capsule form after each meal. Every ten days, 200 roentgen units of filtered superficial x-ray therapy was given by a roentgenologist.

May 3, areas of fluctuation at the angle of the mandible and below the zygoma were aspirated under gas anesthesia. No incision was made.

May 20, the patient was receiving 25 grains of thymol crystals daily without any gastric distress. He was comfortable, with no abscesses forming. Administration of thymol was stopped and potassium iodide was given, beginning with 10 drops and increasing each dose 1 drop, with three doses daily to be increased to 50 drops.

June 1, the disease had become active again and the patient suffered greatly. The right eye was swollen shut, with an area of fluctuation just below and laterally from the orbit. There was also a purulent discharge from the right

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lower third molar socket. The fluctuant area was aspirated under gas anesthesia and the patient was again given thymol. The potassium iodide was discontinued.

June 10, the patient was comfortable, but there was a small fluctuant area over the right zygoma. This was aspirated under gas anesthesia.

Outcome.—July 1, the patient was comfortable. No fluctuant areas were noted.

July 15, all areas had healed, with no evidence of activity. The administration of thymol and x-ray therapy were discontinued.

September 1939, there had been no recurrence. Examination September 1940 was also negative.

CASE 2.—*History.*—J. O., a man, aged 52, a farmer, had had a carious lower right third molar removed one month before. There had been a moderate amount of soreness around the tooth for about a week before its removal. After extraction of the tooth, the discomfort had gradually increased with a gradual swelling along the right angle and horizontal ramus of the mandible.

Examination.—September 27, 1941, examination revealed swelling at the angle and along the right horizontal ramus of the mandible. There was an area of fluctuation just below the angle. The skin over the fluctuant area was a dusky purplish hue. There was no history of putting straw or sticks in the mouth. The jaws were edentulous, with an unhealed lower right third molar socket from which a small amount of purulent material could be expressed. X-ray examination revealed an area of rarefaction extending about 0.5 cm. around the third molar socket. There was no evidence of sequestration.

Treatment and Course.—Under gas anesthesia, incision and drainage were carried out with aspiration of a moderate amount of purulent matter. Sulfur granules were found and microscopic examination confirmed the clinical diagnosis of actinomycosis. The patient was given superficial x-ray therapy by the roentgenologist, with thymol crystals in capsule form, 5 grains after each meal, the dosage increasing each day by 5 grains until 45 grains was being taken each day.

October 7, 1941, there were small areas of fluctuation opposite the body of the ascending ramus of the mandible and along the lower border of the mandible in the mental region. Under gas anesthesia, the purulent discharge from both regions was aspirated into a syringe through a large needle. Thereafter, the patient progressively improved.

Outcome.—October 21, the thymol was decreased to 15 grains daily. No activity of the disease was evident and the patient was comfortable except for trismus.

November 4, x-ray therapy and thymol administration were stopped. December 4, there had been no recurrence. Some trismus remained. January 4 and July 6, 1942, there had been no recurrence and the patient was dismissed.

CONCLUSIONS

1. Thymol crystals are of definite therapeutic value in the treatment of early actinomycosis.

2. Aspiration is as beneficial to the patient as incision and drainage, with much less subsequent scarring.

Medical Arts Building.

AMELOBLASTOMA (ADAMANTINOMA) OF THE MANDIBLE: REPORT OF CASE

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History.—M. P., colored, aged 35, a housekeeper, seen October 17, 1940, first noticed enlargement of the right side of the face and jaw after extraction of a lower right third molar, in March 1939. Following the extraction, there was very severe hemorrhage, which was stopped the following day with considerable difficulty. There was also an increase in the amount of swelling, associated with considerable pain, which was controlled with sedatives. Finally, the patient went to Trinity Hospital in Detroit, in June 1939, and the tumor at that time was excised. The tumor never entirely disappeared after the operation. In February 1940, the patient was seen at the Grace Hospital, Detroit, where another operation was performed, but the tumor was not completely removed.

The patient had had the usual childhood diseases—measles, mumps, etc.—but she had never had a doctor until the onset of the present illness. She had never had any other serious illnesses. She was in an automobile accident in 1936 and sustained minor lacerations of the hand and forehead. Otherwise, the history was not significant. The patient was married, but separated from her husband. There were no children.

Physical Examination.—The patient was well developed and well nourished, with no other complaints than the tumor of the jaw. The blood pressure was 115/60; pulse, 72; temperature, 98° F. The results of a Kahn test were negative. Urine tests were negative. The red blood cell count was 4,900,000; white blood cell count, 8,500, with 75 per cent hemoglobin. The blood type was II.

A large tumor mass involved the entire lower right side of the face and was causing extensive deformity from the midline of the mandible on the right side around to the region of the right temporomandibular joint. Extra-oral palpation revealed a large semisolid, nonfluctuant, tender tumor-mass extending 4-5 cm. below the inferior border of the mandible laterally, causing stretching of the overlying skin, and tumescence involving the area of the angle of the mandible and the ascending ramus on the right side. This mass extended up as far as the zygoma. There was no limitation of motion of the mandible and the entire mass moved on motion of the lower jaw. Intra-orally, this tumor was found to extend from the mandibular right lateral area posteriorly to involve the entire jaw. All of the teeth back of the cuspids had been extracted and there was a large ulcerative tumor mass involving the alveolar ridge. This extended laterally into the cheek and medially through the floor of the mouth, and palpation revealed medial movements of the lateral wall of the pharynx. The tumor was also impinging on the floor of the mouth, pushing the tongue over to the midline. Intra-orally, the mucous membrane overlying the tumor was fixed to the tumor mass, but, extra-orally, the skin overlying the tumor mass was, although tightly stretched, not fixed to the tumor. There was no

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weakness of the muscles of expression on the right side and apparently no involvement of the parotid gland or the seventh nerve.

X-Ray Examination.—Routine films revealed nearly complete destruction of the right half of the mandible. Only very thin walls remained. Large cystic areas extended from the anterior portion of the horizontal ramus up to the condylar process of the mandible. There appeared to be rather marked soft tissue swelling of the right half of the face. (Fig. 2, left.)

Diagnosis.—The diagnosis was adamantinoma of the mandible.

Operation (By the author, assisted by Drs. Kemper, Keihn and Shemiot).—October 25, under intratracheal nitrous oxide and ether anesthesia, an incision was made along the lower border of the tumor from the symphysis posteriorly to the region of the angle of the mandible, and then upward behind the ear on

Fig. 1.—Preoperative photograph.

the right side. The skin, subcutaneous fascia and cervical fascia were incised. The tumor mass was exposed, and, by blunt and sharp dissection, the entire tumor mass was exposed and identified with a moderate degree of difficulty. The mandible was cut through at the symphysis with a Gigli saw. The muscular and periosteal attachments were dissected from the tumor mass and from the small amount of bone that was left in a few areas about the mandible, and the entire tumor, with a small amount of remaining bone, was removed *in toto*, after disarticulation of the right temporomandibular joint. The wound was closed in layers with interrupted ooo chromic sutures. The skin margins were approximated with interrupted silk sutures. Three large Penrose drains were

inserted and sutured to the skin margins. The diagnosis of adamantinoma of the mandible was confirmed. (Figs. 3 and 4.)

Progress.—On return from the operating room, the blood pressure dropped to 80/40. The pulse became rapid and weak. The patient was then placed in shock position, and a blood transfusion of 500 cc. of citrated whole blood was administered. This was followed by 2,000 cc. of intravenous 5 per cent glucose, and, within two hours, the pulse, respiration and blood pressure returned to normal. Thereafter, the postoperative course was entirely uneventful. There was some drainage from the wound, which healed by second intention. The Penrose drains were removed on the fourth postoperative day. The patient was up and about the ward on the tenth day, and was discharged from the hospital completely well, November 9.

Second Admission.—The patient returned to the University Hospital approximately nine months after operation. The only complaint was a noticeable keloid formation along the line of incision of the previous hemiresection of the right side of the mandible. The formation, which was about 1 cm. in width, extended along the full line of incision from the angle of the mandible to the

Fig. 2.—Left: Preoperative roentgenogram, showing tumor. Right: Postoperative roentgenogram; after hemimandibulectomy.

symphysis. Intra-oral examination revealed very good adjustment of the soft tissues to the rather extensive resection. The occlusion of the remaining one-half of the mandible was quite good, and the patient had made a very good adjustment as far as eating was concerned. Laboratory findings were negative and x-ray examination of the chest was negative.

The patient was referred to the department of x-ray therapy, where the keloid was treated with 900 roentgen units. The field of irradiation was limited to the area of the scar. She was discharged from the hospital on the third day after admission, and advised to return for check-up examination in three months.

Third Admission.—The patient was not seen again for approximately one year, returning to the hospital May 5, 1942. She had been perfectly well since her discharge from the hospital in November 1940 with the exception of the development of a keloid, treated at the University Hospital in July 1941, until

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about January 1, 1942. At that time, she first became aware of a return of the swelling about the right side of the face. Since that time, there has been a gradual enlargement of the mass in the region from which the mandible had been resected. Otherwise, the interval history was negative.

Physical Examination.—There was a mass about the size of a baseball in the area from which the right side of the mandible had been resected. The mass was somewhat fluctuant, definitely limited in its outline and rather tender. Oral examination showed that the mass arose along the area of the resected mandible, and it seemed to be attached to the underlying soft tissues in the retromolar area. Oral examination was otherwise negative, and about the same

Fig. 3.—Operative specimen.

as at the time of previous admission. The urine was negative; hemoglobin, 90 per cent; white blood cell count, 9,000. (Fig. 5.)

X-Ray Examination.—Only a few faint areas of calcium density were visible on the side of the resection. The possibility of residual or recurrent neoplasm could not be excluded. The left half of the mandible appeared normal.

Diagnosis.—Recurrent adamantinoma of the mandible was diagnosed.

Operation (By the author).—Under intratracheal anesthesia, an incision

was carried through the old line of scar from the previous resection, and, through this incision, the outline of the tumor was defined and, by means of sharp dissection, the entire tumor mass was excised. The adjacent soft tissues appeared to be somewhat fixed to the capsule of the tumor and, for this reason, it was not possible to remove the tumor by shelling it out of its capsule. It was removed by sharp dissection. In the region of the hyoid bone, there was a definite fixation to the skin and fascia, but the tumor was removed in this area by a rather wide excision. The diagnosis was confirmed.

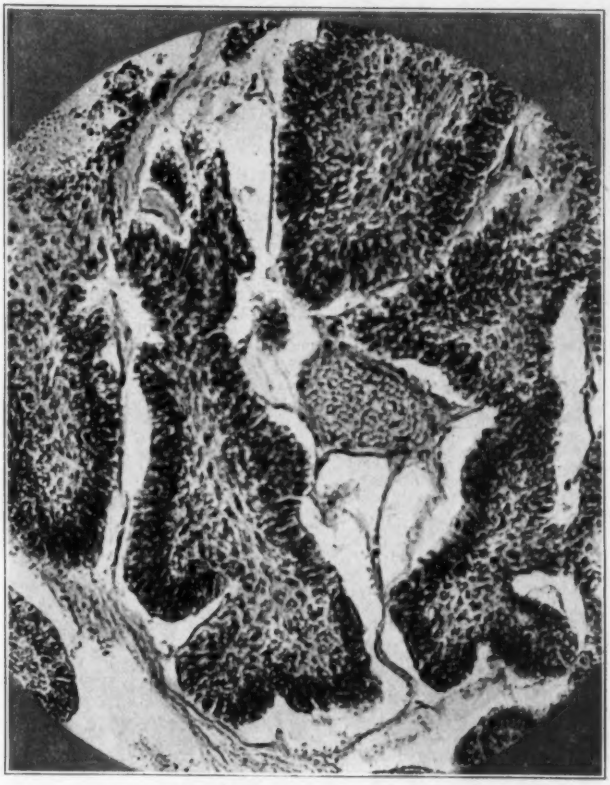

Fig. 4.—Photomicrograph of operative specimen.

Progress.—Postoperatively, the patient was fairly comfortable, but complained of a burning sensation in the mouth. On the fourth postoperative day, when the sutures were removed, it was found that there was a breakdown of about 1 cm. in the line of incision. Two days later, an oral external fistula at the operative site was noted. Warm saline irrigations were given daily, and a small pack was placed in the site of the fistula. Intra-oral and extra-oral dress-

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Fig. 5.—Recurrent adamantinoma, eighteen months after first operation.

Fig. 6.—Final result.

ings were changed daily, and it was noted that the fistula was closing gradually. May 23, the patient was discharged from the hospital with instructions to

return in one week for check-up. The area of incision of the tumor was completely healed, and the fistula at that time had also completely healed. The patient no longer complained of the burning sensation in the mouth and tongue. The oral mucosa in the right side of the mandible was healed.

January 14, 1943, the patient returned for observation. She stated that, during August 1942, she had had pneumonia, with a residual persistent cough. There were no complaints regarding the operative site, and examination revealed the latter to be well healed. (Fig. 6.)

Comment.—This case again illustrates the responsibility of the first surgeon who operates in a case of adamantinoma. He has the best possible chance of curing any tumor. Inasmuch as there is a possibility of recurrence, these tumors should be treated rather radically and completely removed. They will recur if any portion of them is left. Because of the great possibility of recurrence and the malignant potentialities, incomplete surgical measures should have no place in the management of adamantinoma.

Roentgenology in Diagnosis.—We must keep in mind that we are looking at shadows instead of pathologic processes. The shadows which are produced on the film may be evidence of pathologic change in the osseous structure; they may indicate variations of the anatomic structure, or they may be evidence of traumatic changes; all of which must be considered in evaluation of the film or plate. You are all constantly receiving dental films in the mails asking you to make a diagnosis of the case. Some of the films are so indefinite that even a guess that it was a dental film would be hazardous. A diagnosis in such cases can be of little value. The age of the patient, the physical resistance, the possible recuperative powers must all be coupled with the roentgenographic findings before we make the diagnosis. It has long been conceded that in some of the most vicious infections arising from foci in and about the teeth, the roentgenographic evidence is negative. It has also been demonstrated that some patients may for a long time tolerate certain areas of rarefaction about the apices of teeth without apparently any untoward incident. Again, the condition is largely dependent on the physical resistance of the patient. . . . I am merely attempting to show that, as a single means of diagnosis, the dental film falls far short of the value that many men place on it.—Chalmers J. Lyons, in *J.A.D.A.*, 1932.

SECTION ON WAR DENTISTRY

CYSTIC CAVITY IN THE MANDIBLE: REPORT OF A CASE

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IN an Army general hospital, dentistry is recognized as a valuable health service in the medical treatment of the patient. Medical and surgical cases hospitalized for complaints involving a probable focus of infection are referred for dental consultation, which includes complete intra-oral x-ray examination. It is not uncommon to have a roentgenographic survey disclose pathologic processes that could not be observed clinically. Asymptomatic periapical disease, cystic defects and other bone lesions are frequently diagnosed from the x-ray films, although the patient is unaware of the presence of these conditions.

This report is concerned with a cystic defect of the mandible found on dental examination of a patient admitted to the medical service with a chief complaint of arthritis. The cystic cavity was first noted on dental x-ray exposure. Lateral jaw plates were then made to verify the presence and determine the extent of the cavity. The Medical Service elected to have this area of apparent bone disease operated on to rule out a probable source of infection referable to the arthritic complaint. The presence of a cystic lesion in the head of the right femur also suggested the possibility of a polycystic disease. Such lesions as the xanthoma, malignant lesions, giant-cell tumor, multiple myeloma, generalized osteitis fibrosa cystica and cyst of dental origin could be pretty well eliminated by a consideration of the history, physical findings, laboratory reports and roentgen appearance of the cavity.

REPORT OF CASE

History.—A man, colored, aged 45, was admitted to the hospital with a complaint of arthritis of the hip, left leg and left shoulder. The history was essentially negative. The first symptoms of the present illness had been noted about seven months before admission to the hospital.

Orthopedic Examination.—There was some pain, but not much limitation of motion to the left shoulder joint. The patient could not raise the left leg as high as he could the right and movement caused pain in the sacroiliac joints.

X-Ray Examination.—The lumbar spine showed obliteration of the paired cartilages in the upper half on each side, more marked on the right. Bone condensation changes were noted bilaterally. The lumbar spine was normal in the anteroposterior view. The left shoulder joint was negative. A fibrocystic

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area in the right femoral head was noted. Both tibiae and humeri were negative. Chest films were negative.

Dental Examination.—All teeth were vital and the x-rays failed to reveal apical disease. There was moderate to advanced periodontoclasia in the right and left posterior maxillary region. One corner of the cystic defect was disclosed on the lower right molar film. The extra-oral film (Fig. 1) showed a well-circumscribed area of decreased density extending upward from the lower border of the mandible almost to the inferior dental canal. It appeared to be entirely enclosed in bone. This area was ovoid and the walls of the cavity were thicker and more dense than those ordinarily seen in a dental cyst. An x-ray film of the left mandible showed no pathologic process. There was no history of localized trauma or any symptom associated with the cystic cavity. The lesion was reported as a possible source of focal infection, although the probability of its not being pathologic was also recognized.

The laboratory data are given in the accompanying tabulation.

LABORATORY DATA

Test	Normal	Patient's
Blood phosphorus	3- 4 mg.*	3.2 mg.*
Blood calcium	10-12 mg.	10.3 mg.
Uric acid	2- 4 mg.	3.8 mg.
Non-protein nitrogen	20-35 mg.	34.8 mg.
Kahn	Negative	Negative
Urinalysis	Negative	Negative
Basal metabolism	+10 to −10	−20%
Sedimentation rate	0-15 mm. drop at end of one hour	4 mm. drop at end of 1 hour
Blood count		5,090,000 red cells 5,950 white cells Polymorphonuclears 56% Lymphocytes 40%

*Per hundred cubic centimeters.

Preoperative Diagnosis.—Cyst, type undetermined, was diagnosed.

Operation.—Under endotracheal ether anesthesia, an incision 1½ inches long was made beneath the shadow line of the mandible near the angle, extending forward. The muscle tissue was carefully dissected and the external maxillary artery and vein were located, ligated and cut. Further dissection through the muscle tissue was carried out to expose the periosteum and bone of the mandible. The bone defect was found to interrupt the continuity of the lower border of the mandible. The periosteum over the bone was reflected back and a small particle of apparently normal, but dense cortical bone making up the lateral wall of the cavity was removed with rongeur forceps. This defect had no contents, no fibrous membrane and no epithelial lining. Sulfanilamide powder was dusted into the area and the periosteum and muscle tissues were closed in layers with 000 chromic sutures. The skin was closed with interrupted sutures. A small drain was inserted at the posterior end of the incision and a pressure gauze dressing applied.

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Postoperative Course.—The postoperative course was uneventful. The sutures were removed on the fourth and fifth days. A slight serous discharge ceased after eight days. Figure 2 shows the healed incision two weeks after operation.

Pathologic Report.—The specimen consisted of a small particle of cortical bone showing no gross abnormality. Microsection revealed a small portion of normal cortical bone.

COMMENT

It is rather obvious that we were dealing with a non-pathologic bone cavity. Although the defect involved the lower border of the mandible, it was not palpated during the examination because of the obesity of the patient and the fact that it was located at the slight depression in the bone where the external maxillary artery and vein cross the mandible. There can be no correlation between this cavity and the other complaints of the

Fig. 1.—Right lateral jaw; showing cystic defect, which appears to be entirely enclosed in bone.

patient. The results of the surgical and microscopic findings best explain this bone cavity of the mandible as of congenital or embryologic origin. This serves to collaborate the theory of Stafne,[1] who suggests that this condition may be a definite entity and may be congenital.

Stafne states that it was possible that a cavity could result and persist from failure of normal bone deposition in a region formerly occupied by cartilage. He reported thirty-five cases of cystic cavities of the mandible, one of which was bilateral. Most of the defects in his series were confined to the cancellous structure of bone, but those of greater size involved the lower border of the mandible.

The formation of the mandible from Meckel's cartilage, accessory centers

such as the condylar and coronoid cartilages and membranous bone is a complex affair, and one might expect cyst formation. As development progresses, these cartilaginous centers become enclosed in bone and the cartilage is squeezed out of existence. A failure of bone deposition to replace cartilage and, later, enlargement and liquefaction of old embryonic cartilage would lead to bone absorption and cavity formation.

The location of this cystic cavity in the mandible might also suggest the possibility of another embryologic anomaly as the cause. The external maxillary vessels cross the mandible in close proximity to this region, and often the bone cavities are close to or in contact with the inferior dental canal. There may well have been some aberrant vessel present during the embryologic development of the jaw, which later disappeared, leaving the cavity in the bone. In either case, the bone cavity results from an aplasia rather than dysplasia of the formative elements. The conditions necessary

Fig. 2.—Healed incision, two weeks after operation. A small drain had been placed at the posterior end of the incision.

for the formation of normal bone were present, but an extraneous element existed which prevented normal development.

I believe that these bone cavities are more common than is apparent from a review of the literature, but because of a lack of symptoms, they have not received much consideration. Many such cavities may well have been interpreted as traumatic cysts. Havens[2] included four cysts of undetermined origin in a report of 198 dental cysts. These were located near the angle of the mandible. Three cysts had no epithelial lining, but did contain a gruel-like material, and were explained by the author as being of traumatic origin. The fourth cyst had a stratified squamous epithelial lining and was considered to be of dental origin. However, it is rather difficult

to explain cysts lying entirely below the inferior dental canal as of dental origin. Penhale[3] reported a lesion similar to the cavity described in this paper. His diagnosis was traumatic cyst, although there was no history of trauma associated with the lesion.

The so-called traumatic cyst was first described in this country by Blum and the pathology presented by Thoma.[4] These cysts have no epithelial lining, but usually contain decomposed blood elements and serous transudate. Surgical treatment is necessary to obliterate the cyst as it may increase in size and gradually involve and destroy more bone.

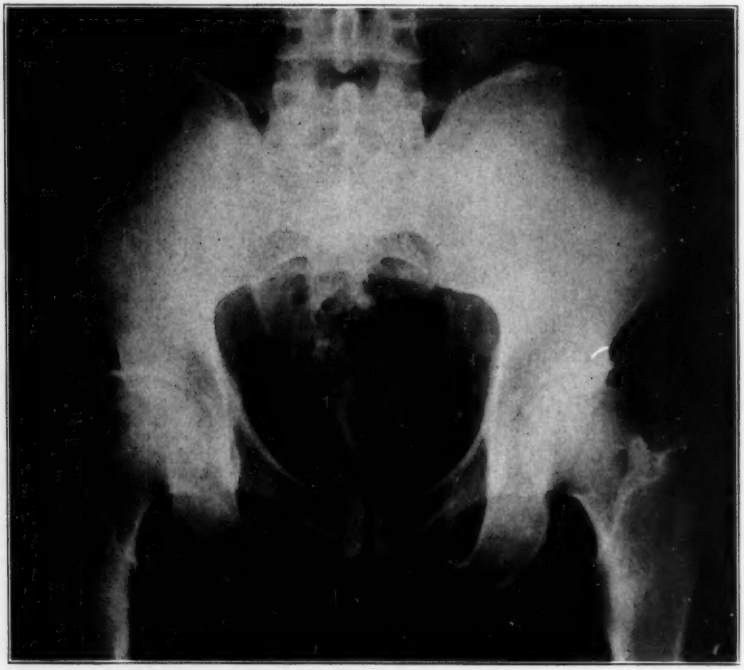

Fig. 3.—Cystic lesion in right femur. The exact nature and cause of the cyst were unknown.

The bone cavity formed in the course of embryologic development requires only periodic observation. This is necessary primarily to substantiate the diagnosis. Other cysts of the mandible that may be pathologic would show changes in size and appearance over a period of time. The congenital defect could hardly be expected to show any change after growth of the mandible ceases. Stafne reported no change in the x-ray appearance during a five to ten year interval in some of his cases.

The fibrocystic area described in the femoral head could well be of congenital origin, although the exact cause of these solitary cystic lesions in the long bones is unknown. In explaining the presence of these lesions in the long bones, Phemister and Gordon offer four main hypotheses: They may result from degenerated tumor, localized fibrocystic disease, a hematoma in the bone marrow or infection. Copeland and Schickter[6] state that, in 78 per cent of cases, lesions in the long bones were healed fibrocystic areas responding to treatment of hyperparathyroidism. Witting and Gillespie[7] reported a case of benign solitary bone cyst in the fibula of a newborn child. This lesion healed spontaneously. The possibility of an endochondral pathosis, as described by Ollier, could be eliminated in this case by the surgical and microscopic findings. This disease is a result of dysplasia rather than aplasia of cartilaginous elements, which leads to cavity formation in bone.

CONCLUSIONS

1. There are bone cavities near the angle of the mandible which are non-pathologic and not of traumatic origin, but probably congenital.

2. These cavities can be checked periodically by x-ray examination as a possible means of differential diagnosis. Surgical intervention is not indicated.

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INCISIVE CANAL CYST OF THE MAXILLA

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INCISIVE canal cysts are not particularly common, if we can judge by the literature. No doubt, many existing incisive canal cysts have not been diagnosed owing to an absence of symptoms, being discovered during a routine roentgenographic examination of the teeth. It is presumed that, in many cases, the cyst has been regarded as an unusually large nasopalatine canal and promptly ignored.

Palatal cleft cysts have been described by Grüenwald. (Thoma.) They have also been described by Schroff, as nasopalatine duct cysts. In the adult, the nasopalatine duct occasionally remains open or the lumen of the duct closes, leaving an impervious cord of epithelium. Usually, however, it is completely obliterated.[1]

The etiologic factor in the formation of a cyst in this region is possibly trauma, irritation of the papilla or nasal infection. It is formed from the nasopalatine duct, either at the foramen or in the incisive canal.

Mead's "Oral Surgery" quotes Myer as saying that probably most of them arise from the epithelial remnants left from the time of formation of the palate.[2]

These incisive canal cysts are not similar to the fissural cysts or the radicular cyst and are not dental in origin. The patient usually reports symptoms of pain, pressure and swelling or complains of pressure pains of the teeth or of the nose. The patient may also complain of difficulty in breathing. The cysts contain the characteristic cystic fluid, which may be straw or amber colored, and may vary as to consistency. They may also be complicated by secondary infection.

REPORT OF CASE

History.—A man, aged 40, was admitted to hospital December 1943 because of persistent pain in the upper jaw and swelling of the palate with intermittent discharge. He was first seen aboard the *U.S.S.*———— in September 1943 because of the same complaints, which had been present for the previous six months. Examination in March 1943 revealed a localized swelling in the vault of his palate. This was incised and drained aboard the *U.S.S.*———— approximately one month later. The patient stated that he was told at the time that the mass was a cyst.

The mass reappeared in May 1943 and had since slowly enlarged. It was again incised and drained in August 1943 and the cavity was packed with gauze. Since that time, there had been a persistent dull ache in the upper jaw.

The patient had served continuously in the Navy for twenty-one years, dur-

*Lieutenant Commander (DC) V-(S), USNR.

ing which time he had been treated for syphilis and gonorrhea. He had had a tonsillectomy and a mastoidectomy, complicated by brain abscess, during that period. Physical examination at the time of entering the hospital revealed a discharging fistula at the point of the nasopalatine foramen. The fluid was serous and a light straw color.

Examination.—Roentgenographic examination revealed a large well-circumscribed area extending from the midline of the maxilla approximately 20 mm. laterally and 28 mm. in depth. (Fig. 1.)

Vitality tests of the upper central incisors were negative.

The remainder of the physical examination was essentially negative. The diagnosis of incisive canal cyst of the maxilla was made and the following procedure carried out.

Fig. 1.—Condition before operation.

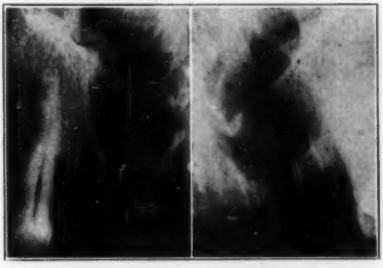

Fig. 2.—Condition after operation.

Operation.—Under local anesthesia, a semilunar incision was made, extending from the second bicuspid on the right side to the second bicuspid on the left side. When the palatal flap was retracted, destruction of the palatal bone in the area of the incisive foramen was revealed.

The cystic lining, which was firmly attached to the bone, was carefully enucleated by blunt dissection. The resulting cavity extended to the wall of the left maxillary sinus and had completely destroyed the bone of the floor of the nose.

There was severe hemorrhage from the palatine artery, which was thereupon

tightly packed with gauze, an extension of the packing acting as a wick being brought down to the surface. The flap was brought into position and sutured with interrupted ooo dermal sutures. The upper central devitalized teeth were removed at the time of operation, but it is to be noted that the devitalization and removal of these teeth were in no way related to formation of the cyst.

Course.—The sutures and packing were removed on the fourth day. Healing was uneventful and the patient was discharged to duty ten days after the operation. (Fig. 2.)

SUMMARY

In a case of incisive canal cyst, incision with drainage was inadequate, only complete enucleation offering a permanent cure.

It is suggested that in all questionable enlargements of the incisive canal, aspiration be employed to differentiate between an incisive canal cyst and a simple incisive canal enlargement.

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The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval service at large.

Cysts.—The simple cyst sometimes referred to as the radicular cyst is quite a common lesion. It is nearly always associated with pulpless teeth or tooth roots and is frequently found in edentulous jaws long after such a tooth or tooth roots have been extracted. Just how and why these cysts develop has long been questionable and is not at this time clearly established. They probably have their beginning in the so-called granuloma, but why some of the granulomas develop into cysts while others do not is obscure. It seems to be a clinical fact that certain of these so-called granulomas increase in size until a large cavity in the bone is formed. The contents of the granuloma become fluid in character and undergo degeneration. The fluid increases in volume as the cyst develops and the bone immediately surrounding the cyst is gradually resorbed by pressure resorption until a large area is destroyed. These cysts are lined with an epithelial membrane. Just where this epithelial lining comes from is largely speculative. Many investigators believe that it develops from a peridental epithelial remnant.—Chalmers J. Lyons, in *J.A.D.A.*, 1928.

Book Review

ATLAS OF THE MOUTH AND ADJACENT PARTS IN HEALTH AND DISEASE. Prepared by Maury Massler, D.D.S., M.S., Director of the Child Research Clinic, Assistant Professor of Histology and Lecturer in Stomatology (College of Medicine), and Isaac Schour, D.D.S., Ph.D., D.Sc., Professor of Histology and Head of the Department of Histology, University of Illinois, College of Dentistry. Drawings by Carl T. Linden. 180 illustrations (19 in color). Published and distributed by the Bureau of Public Relations and the Council on Dental Health, American Dental Association, Chicago, Ill., 1944. Price $2.50.

This exceptionally well-illustrated atlas presents in compact form essential applied anatomy of the oral cavity and adjacent regions in health and disease. Throughout the book, brief and excellent text matter faces the accompanying plate and each turn of the page introduces a new subject. The first portion of the book, which is devoted to the teeth, covers the permanent and deciduous arches, relationship of enamel dentin and pulp, normal and abnormal occlusion, tooth development, physiologic spacing, dental caries, the effects of loss of teeth, erosion, dental fluorosis, chronology of tooth development, growth and calcification patterns, enamel hypoplasia and the teeth in congenital syphilis. These conditions are of great importance to the dentist in his every day practice.

Excellent color plates show common lesions of the buccal mucosa. The progress of periodontal disease, as well as oral disease of occupational origin, is well depicted. Plates 24 to 49 are of particular interest to the oral surgeon as well as the general practitioner of dentistry. The topographic anatomy, benign lesions and systemic disturbances of the tongue are well presented. Excellent illustrations of the floor of the mouth, the salivary glands and ducts are included.

The anatomy and common ailments of the throat and the paranasal sinuses are considered and the effects of mouth breathing are well illustrated and explained. Plates of frontal and horizontal sections of the face are accompanied by text covering the pathways of extension of dental infection.

Plates of the arteries, veins and nerves of the head should be found most helpful. An excellent discussion of cavernous sinus infection and thrombosis of dental origin is included.

The anatomy, function and abnormalities of the temporomandibular joints are well illustrated.

Five plates showing the muscles of mastication are excellent forerunners for five plates covering the various types and combinations of fractures of the lower jaw.

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A MESSAGE FROM THE PRESIDENT THE FIFTH WAR LOAN DRIVE

The Treasury Department will soon announce the Fifth War Loan Drive and appeals to all members of the dental profession to respond even more generously than they did for the Fourth War Loan.

The Loan Drive will probably start on Flag Day, June 14, and end on Independence Day, July 4. These days are significant in American History and should inspire each of us to buy another bond as an investment, and as another evidence of our continued support for Victory.

I hope that all component societies will put their efforts behind the Fifth War Loan Drive with the same enthusiasm that characterized the dental profession in the Fourth Loan. Let us purchase more bonds—especially between June 14 and July 4.

C. RAYMOND WELLS,
President.

BACK ISSUES WANTED

The supply of the January 1943 and the April 1943 issues of the JOURNAL OF ORAL SURGERY are exhausted. Many libraries of dental societies, schools and hospitals did not subscribe in time and now wish to make their files complete. The JOURNAL would appreciate receiving copies of these issues from subscribers who can spare them. Returned copies will be paid for or the subscriptions of those subscribers returning them will be extended by one or two issues, the extension depending on the number of copies returned.

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